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SURVIVAL IN PRIMARY CARCINOMA OF THE LUNG*

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THE ability to utilize a lung as a unit of excision has made it possible to think in terms of cure when that organ is the primary site of cancer. This state of affairs has existed but for fifteen years,[1-4] and before that time cancer of the lung was invariably fatal. However new the experience in the surgical management of cancer in this location, a sufficient period has elapsed to permit a fair appraisal of benefits in respect to palliation and cure rates.

During the period in which surgery has been applied for carcinoma of the lung, one of us (R. H. O.) and his associates have examined a total of 604 patients in whom the diagnosis of primary carcinoma of the lung was made. This report is concerned with the ultimate fate of these patients. Survival periods in relation to clinical and operative findings, pathology and the extent of resection are given below. The factor of time between the onset of symptoms and the first request for help and the duration of the period of observation before reference for possible surgical treatment are considered. The necessity of setting ahead the time schedule of discovery by the use of screening methods is discussed.

The recent medical literature contains a number of comprehensive accounts that deal with incidence, symptoms and clinical manifestations of the disease, as well as with technical matters of lung excision.[5-8] The over-all problem of cancer of the lung has been particularly well presented by Churchill.[9] A discussion of clinical features is not included in this report.

MATERIAL

From June, 1932, to August, 1948, a total of 604 patients in whom the diagnosis of primary carcinoma of the lung was made were seen. In 481 cases, the diagnosis was supported by microscopical proof. Great care has been taken to include only cases of carcinoma. Dr. Shields Warren and his associates have again reviewed the histologic sections and have excluded all cases of so-called "malignant" adenoma, adenoma, lymphoma and all secondary growths. In the unverified group no case was included in which there was reasonable doubt of the diagnosis. It is necessary to consider all cases seen if a true picture of operability and over-all salvage rates is to be obtained.

The sex incidence in this series parallels that in other reported groups. Of the verified cases, 85.8 per cent were in males and 14.2 per cent in females.

OPERABILITY

It has been our practice to advise surgical exploration in all suspected or proved cases with but two exceptions: absolute or strong presumptive evidence that the growth is out of bounds; and cases in which the patient's general condition is too precarious or there are complicating factors such that there would not be a reasonable chance of success with either exploration or excision (Table 1).

TABLE 1. *Data in 604 Cases of Primary Carcinoma of the Lung (1932–1948).**

OPERATION		NO. OF CASES	NO. OF DEATHS
Exploration only		127	15 (11.8 %)
Resection:		162	29 (17.9 %)
Without extrapulmonary extension	55		
Microscopical extension only	49		
Gross extension	58		
Totals		289	44 (15.2 %)

*Of these, 481 were verified.

TABLE 2. *Data in 234 Cases of Primary Carcinoma of the Lung (1932–1943).**

OPERATION		NO. OF CASES	NO. OF DEATHS†
Exploration		58	9 (15.5 %)
Resection:		41	9 (21.9 %)
Without extrapulmonary extension	20		
Lymph-node extension only	10		
Gross extension	11		
Totals		99	18 (18.1 %)

*Of these, 190 were verified.

†The five-year survival (4.3 per cent of all cases seen and 5.3 per cent of all verified cases) was distributed as follows: 8 cases without extrapulmonary extension and 2 cases with lymph-node extension only.

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Of all cases, 48 per cent were explored, and resection was carried out in 27 per cent. In recent years, there has been an encouraging trend toward higher rates of operability, and in the past five years, 51 per cent of all cases seen have been explored and 32 per cent resected.

In Table 1 and 2 and Figure 2, 3 and 4 the resections are divided into three categories according to the extent of the lesion: patients with no demonstrable extrapulmonary extension (these are the best candidates for cure); those with lymph-node metastases only, the regional lymph nodes being grossly involved in some and the metastases being found on microscopical examination in others (candidates for possible cure if the excised nodes represented the sole metastatic process); and those with gross evidence of extension, in turn divided into patients in whom all tumor was thought to have been removed (candidates for cure if all tumor is removed) and those in whom gross tumor was left behind (palliative procedure only).

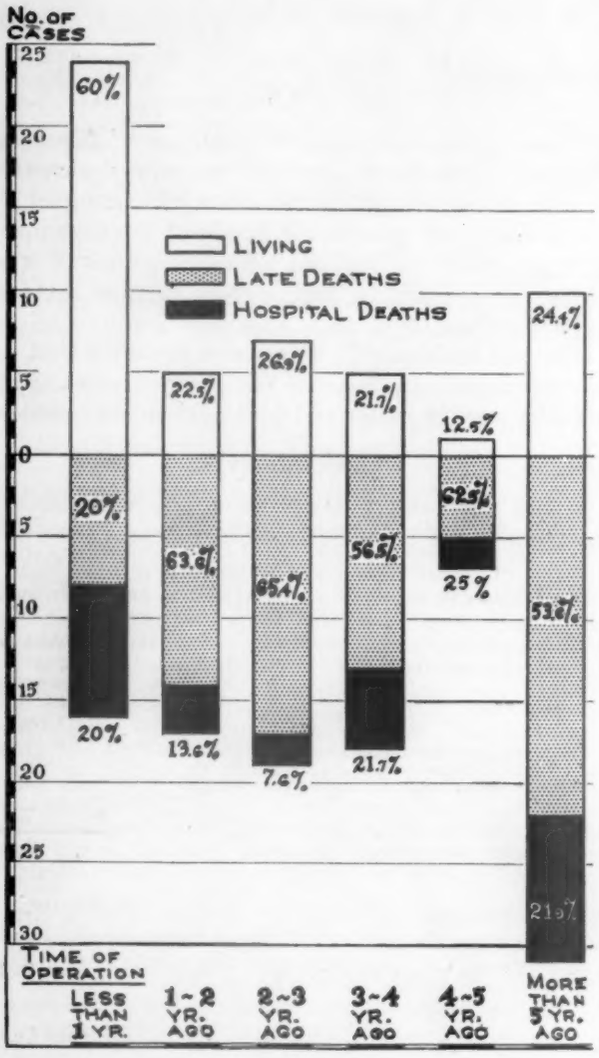

FIGURE 1. *Survival in Total of 162 Patients who Submitted to Resection.*

There were 29 operative deaths, 2 patients who could not be followed, 3 who died of causes other than recurrent cancer, and 2 epidermoid carcinomas that were not graded (leaving a total of 126 cases, which are considered in the succeeding figures).

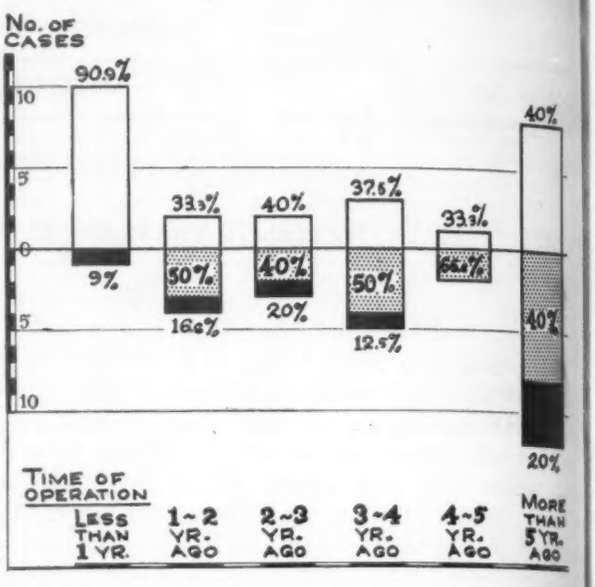

FIGURE 2. *Survival after Resection in 56 Cases without Extension.*

Survival Periods

The survival periods of patients who were known to have died from their disease are shown in Table 3.

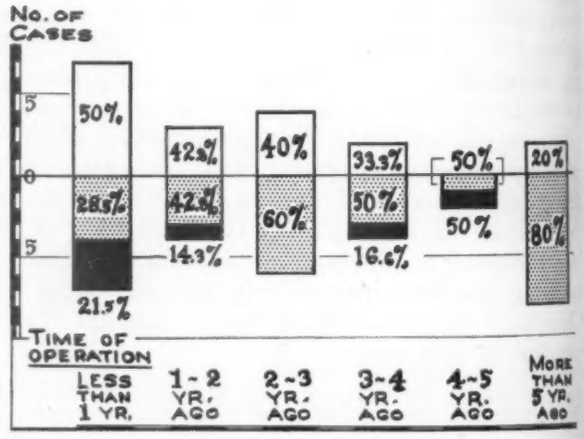

FIGURE 3. *Survival after Resection in 49 Cases with Lymph-Node Extension Only.*

In cases in which the cancer was obviously out of bounds and in which no surgical treatment was offered the patients lived an average of four and two-fifths months. At the other end was a group of 19 patients submitted to resection in whom the growth apparently was localized but who subsequently died of their disease. The average survival of this group was eleven and three-tenths months.

Figure 1 shows the survival periods of all patients who submitted to pulmonary resection. It will be seen that of the patients operated upon within the past year, 60 per cent are still alive, with 8 late deaths and 8 operative deaths. Of those operated upon five years or more ago, 24.4 per cent are still alive (10 patients).

An analysis of the survival periods in the cases without extension is shown in Figure 2. It will be seen that of 11 patients operated upon in the past year, there was 1 operative death, and all the rest are still living. Sixteen patients are living more than a year, 14 more than two years, 12 more than three years, 9 more than four years, and 8 more than five years after operation.

If operative deaths are excluded, 10 patients, or 100 per cent of those who survived operation within the past year, are still living, whereas 8 (50 per cent)

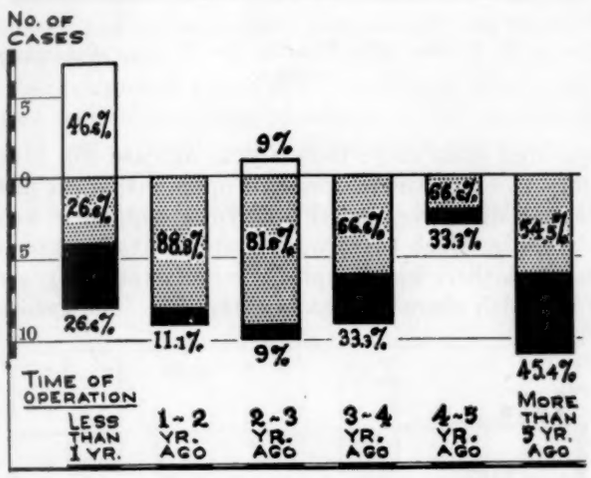

FIGURE 4. *Survival after Resection in 58 Cases with Gross Extension.*

of those who survived operation five or more years ago are still alive.

The average duration of life of those who died postoperatively (excluding hospital deaths) was eleven and one-tenth months. Of these, none lived longer than twenty-eight months.

Cases in which there was lymph-node extension only are shown in Figure 3. In the majority of these cases, the lymph nodes were found to be involved as a result of microscopical study, but in the few cases in which the glands were grossly involved, all tumor tissue was thought to have been removed. As can be seen from the chart, 8 of 28 patients are alive more than two years and 2 of 10 are alive five years or more after operation. Of the late deaths, the average duration of life was nine and three-fifths months. Of these, no patient lived longer than twenty-three months.

The group in which there was gross extension is shown in Figure 4. There were 34 cases in this group in which a palliative resection was done — that is, one in which gross tumor tissue was left behind. In the remaining 24 cases, gross extension was encountered, but all detectable tumor tissue was removed. This group includes the following: cases in which there was invasion of the ribs or chest wall; cases in which there was mediastinal invasion, including those in which there was invasion of the pericardium; and cases in which there was invasion of the diaphragm. In all, the invasion was apparent on gross examination, as distinguished from the lymphatic metastases of the preceding group.

Of 43 patients in this category operated upon more than a year ago, only 1 is alive. This man has an epidermoid carcinoma (Grade II) and has lived

TABLE 3. *Survival Periods from Diagnosis to Death from Disease in Cases of Primary Carcinoma of the Lung.*

Treatment	No. of Cases	Survival
		mo.
None	148	4.4
Exploration	102	7.0
Resection:		
Gross extension	33	10.0
Lymph nodes involved	22	9.6
No apparent extension	19	11.3

more than two years. His was a palliative resection in which gross tumor was left behind, so that there is no possibility of cure. All the other 7 survivors were operated upon less than a year ago. In this group, the average life of those who died postoperatively was ten months, excluding hospital deaths. The longest survivor lived twenty-nine months. This experience indicates that the prognosis is poor if gross extension has occurred, even if all detectable tumor is removed. We have been agreeably surprised at the number of survivors in the group with lymph-node extension. Other reports from the literature bearing upon this subject are few. Ochsner, DeBakey and Dixon[8] reported that in their series of cases with either lymph-node or gross extension, only 2 of 27 patients in this combined group survived beyond a three-year period, and none beyond six years. Coleman[10] reported the cures of 2 patients still living two and six years respectively after resection in which the tumor had invaded the ribs. In our own series there were 6 cases, but no patient has survived more than twenty-seven months.

Survival in Relation to Type of Procedure

The vast majority of patients in our resection series have been treated by pneumonectomy. Only by mass removal of the lung can all hilar lymph nodes be resected, and mediastinal dissection is

facilitated. Occasionally, however, in a patient of advanced age or one with a narrow respiratory reserve who has a peripheral tumor, lobectomy may be preferable. This is particularly true if there is positive evidence of gross extrapulmonary extension. Palliative lobectomy was performed upon a few patients who had peripheral tumors with invasion of the chest wall that could not be totally resected. However, it has been found that these procedures are rarely satisfactory in relieving pain even if coupled with division of the intercostal nerves. Treatment by rhizotomy, high chordotomy or frontal lobotomy if the pain is severe should receive consideration. Lobectomy was performed on 8 patients in our series, 4 of whom were operated upon more than two years ago, and all these are dead. Four patients treated by lobectomy within the past two years are still living.

Pathology in Relation to Survival

The pathological cell type is second only to the extension of the tumor in determining survival. The pathologist has divided all the pathologic cell types into three major classifications: undifferentiated carcinoma; carcinoma arising from glandular epithelium, including adenocarcinoma and carcinoma

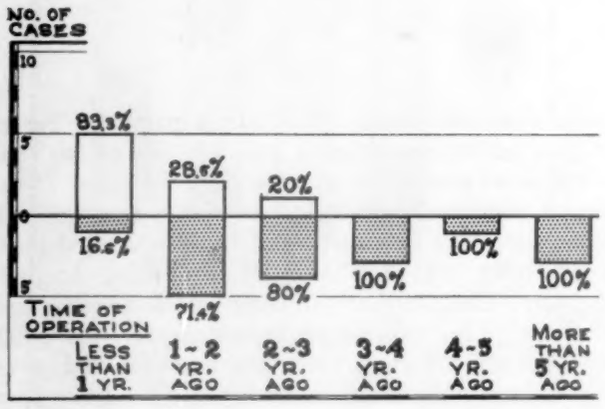

Figure 5. *Survival after Resection in 25 Cases of Undifferentiated Carcinoma.*

simplex; and epidermoid carcinoma, including subgroupings Grades I, II and III.

Only those who survived operation are considered.

In the undifferentiated group (Fig. 5), there were 25 cases. There was no apparent extension of the tumor in 4 of the 7 patients operated upon more than three years ago, and yet none are living. In the late deaths, no patient lived longer than twenty-nine months, and the average duration of life of those who died was nine and three-tenths months.

In the adenocarcinoma group (Fig. 6), there were 12 cases. Of the 6 patients upon whom operation was performed more than three years previously, it will be seen that only 1 patient has survived. In this group of 6 cases, there were 3 without extension, 1 with lymph-node metastases only, and 2 with gross extension. The surviving patient had no demonstrable extension at the time of operation. The average duration of life of those who died was ten and two-tenths months, and of this group, none lived longer than twenty-seven months.

There were 10 cases in which carcinoma simplex was diagnosed (Fig. 7). None of the patients

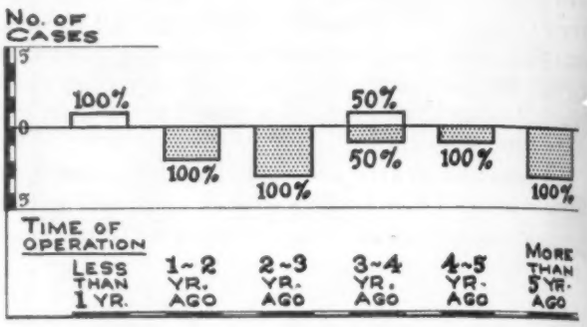

Figure 6. *Survival after Resection in 12 Cases of Adenocarcinoma.*

operated upon more than a year ago are still alive. Only 1 of 3 patients operated upon within the past year is still alive. In this entire group, there were 3 cases in which there was no apparent extension, 4 in which there was lymph-node metastasis only, and 3 in which there was gross extension. The average

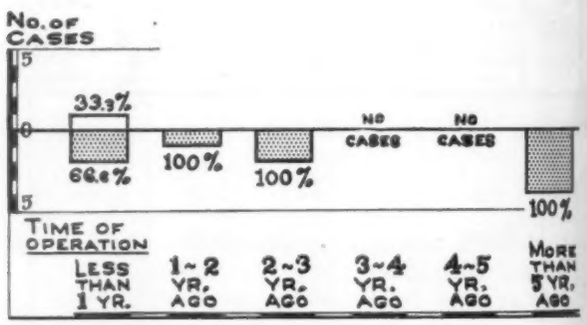

Figure 7. *Survival after Resection in 10 Cases of Carcinoma Simplex.*

duration of life of patients who died was seven and three-tenths months, and none lived longer than thirteen months.

The final group to be considered in relation of survival to pathological type is that in which the diagnosis was epidermoid carcinoma, and it is in these cases that the prognosis is most favorable.

In cases diagnosed as Grade I (Fig. 8) there were 10 cases. It will be seen that 4 of these patients are still living more than two years, 3 of 8 patients more than three years, and 2 of 5 patients more than ten years after operation. The 2 patients living more than five years after operation were both in the group

without apparent extension of any kind. The patients surviving for three and two years both had lymph-node metastases. Of those who died, 4 had gross extension, and 2 had lymph-node metastases; the average survival was fifteen and one-tenth

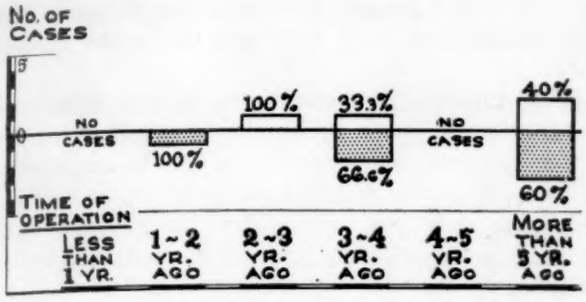

FIGURE 8. *Survival after Resection in 10 Cases of Epidermoid Carcinoma (Grade I).*

months, and yet no patient lived more than twenty-seven months.

In the cases of epidermoid carcinoma (Grade II) there were 36 patients, of whom 20 still survive, 8 of 18 having lived longer than three years, and 5 of 10 having lived longer than five years (Fig. 9). In 3 of 5 cases in which there was no extension of

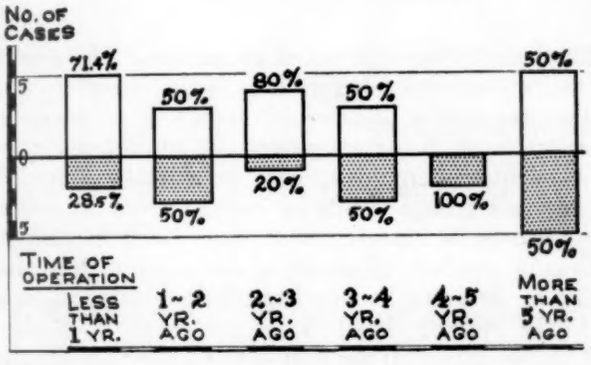

FIGURE 9. *Survival after Resection in 36 Cases of Epidermoid Carcinoma (Grade II).*

the tumor the patients have survived five years or more after operation, and 5 of 9 patients in this same group have survived three years or more. Two of 5 patients with lymph-node metastases are still living five years or more, and 3 of 8 cases are living three years or more after operation. No patient who died lived longer than twenty-four months, and the average survival was twelve and nine-tenths months.

Of 33 cases in the group diagnosed epidermoid carcinoma (Grade III), 3 of 6 patients operated upon five years or more ago and 4 of 8 operated upon more than four years ago still survive (Fig. 10). However, there are no survivors among the patients operated on three, two and one years ago. Of those operated upon more than four years ago, the 4 survivors were in the group without extension of the tumor. In the intervening years, however, 3 other patients without extension have died. Four patients with lymph-node extension upon whom operation was performed more than a year ago have all died, as have 6 with gross extension. The average duration of life in the late deaths was eight and two-fifths months, and no patient lived longer than twenty-eight months.

Symptoms and Survival

All patients submitted to resection were divided into three groups: those who had symptoms for less

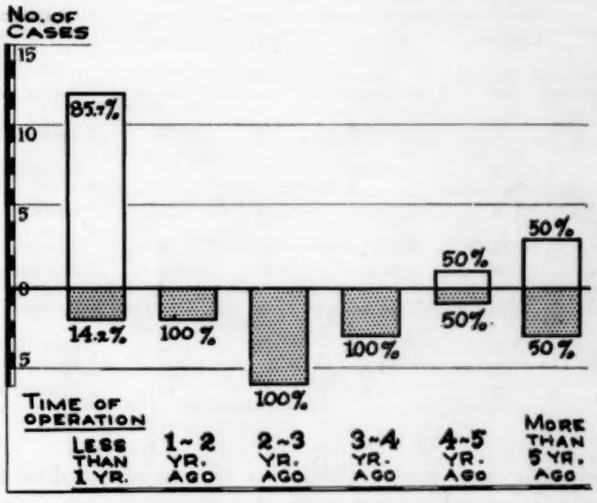

FIGURE 10. *Survival after Resection in 33 Cases of Epidermoid Carcinoma (Grade III).*

than six months; those with symptoms for six to twelve months; and those with symptoms for more than a year. One would expect to find a higher number of survivors in those patients who had symptoms for less than six months, and yet it was found that in this group the percentage of survivors was lower than that in patients who had symptoms for six to twelve months. The percentage of survivors of those who had symptoms for more than a year was about the same as that in patients who had symptoms for less than six months. This finding could be interpreted to mean that many growths are relatively far advanced by the time symptoms develop. No attempt was made, however, to correlate pathology with the duration of symptoms. Many patients with anaplastic tumors presented themselves before they had had symptoms for a year, whereas others with more slowly growing tumors waited longer before consulting a physician.

Those patients who survived for more than five years are shown in Table 4. All were treated by total pneumonectomy. Of these, 5 are well, 2 are reasonably well, and the last available information concerning another revealed that she was well in

1947. Of the 2 remaining patients, 1 was well for ten years but has gone downhill in the past two years and is now a respiratory invalid; the other was well for five years and has had a similar downhill course in the past two years. Both are apparently free of cancer but are suffering from a lack of respiratory reserve.

In the group of patients operated upon more than five years ago, there is an additional patient who died in an accident thirty-nine months after resection. He was working every day until his death, and was apparently free of disease. He presumably had been cured, for in our entire series no patient who later died of his disease has lived longer than thirty months after diagnosis. This includes untreated patients, those in whom thoracotomy only was performed, and those who had pulmonary resection. All who died had signs and symptoms of cancer for several months prior to death, so that it appears from our experience that any patient who lives thirty months or more without symptoms of recurrence has an excellent chance for cure.

TABLE 4. *Results in Patients Who Have Survived Five or More Years after Pulmonary Resection for Carcinoma of the Lung.*

CASE NO.	DATE OF OPERATION	AGE	SEX	FINAL DIAGNOSIS	PRESENT STATUS
		yr.			
1	5/13/36	45	M	Epidermoid carcinoma (Grade II)	Patient well for 10 years; downhill in past 2 years; now respiratory invalid.
2	6/10/37	45	F	Epidermoid carcinoma (Grade III)	According to last information (1947), patient well
3	3/18/41	44	M	Epidermoid carcinoma (Grade III)	Patient well for 5 years; downhill in past 2 years; now respiratory invalid.
4	10/11/41	43	M	Epidermoid carcinoma (Grade I)	Patient well; does light work; some dyspnea upon exertion only.
5	5/7/42	56	M	Epidermoid carcinoma (Grade II)	Patient well
6	4/16/42	48	M	Epidermoid carcinoma (Grade II)	Patient well; works every day; some dyspnea upon exertion only.
7	7/31/42	48	M	Epidermoid carcinoma (Grade I)	Patient fairly well; does light work; tires easily.
8	8/28/42	54	M	Epidermoid carcinoma (Grade II)	Patient well
9	6/29/43	62	M	Epidermoid carcinoma (Grade II)	Patient well
10	7/13/43	55	M	Epidermoid carcinoma (Grade III)	Patient well

FUTURE POSSIBILITIES

The present method of waiting for symptoms, followed by the delay that seems to be necessary to work out the differential diagnosis, has not produced the results that we believe are possible in the present-day surgical treatment of primary carcinoma of the lung.

It was previously pointed out that an analysis of the histories of 133 patients seen between 1932 and 1942 revealed that there was an average interval of eleven and three-fourths months between the first symptom and the establishment of the diagnosis.[11] The average patient delayed seeing a doctor for three months. The first x-ray examination was not ordered until another three months had elapsed. The diagnosis was established five and three-fourths months later. A review of the histories of patients seen in 1947 and 1948 showed that the interval between the first symptom and the date of diagnosis had been reduced only to ten months. The average patient delayed seeing a doctor for three and eight-tenths months. The first x-ray film was ordered one and six-tenths months later, compared with an interval of three months in the earlier group. However, the diagnosis was not established until four and six-tenths months later.

The interval between onset of symptoms and diagnosis will have to be shortened; in fact, this interval can be eliminated altogether. The growth can be discovered before it is large enough to produce symptoms. Radiologic screening of the population, now being employed in uncovering cases of silent tuberculosis, can be further extended. Older age groups should be included, and the screening should be repeated each year. Early carcinomas usually produce changes in the x-ray picture that can be detected by the experienced observer, even if the tumor itself will not cast a shadow. Approximately 80 per cent of the lesions are situated in a lobar or segmental bronchus, and the secondary changes in the corresponding segment or lobe, incident to partial or total bronchial occlusion, cast abnormal shadows detectable in the miniature film used for screening. There is a period during the growth of the tumor when abnormal x-ray densities develop and then symptoms appear. Part of this period of silent growth can be saved. The minority group of peripheral tumors cast direct shadows, which, even in their early stages, should be caught in the screening process owing to the highly rarefied normal lung field. If abnormal densities are discovered, they will require proper identification without delay. Additional x-ray studies should be arranged for immediately. Cytologic examination of the sputum and of material aspirated from the bronchi (Papanicolaou technic),

bronchoscopy or even exploratory thoracotomy may be necessary to settle the diagnosis.

Efforts to discover primary carcinoma of the lung in its silent and early stage will be rewarded. The risk of resection is reasonably low and is constantly being lowered. Five-year survival rates for patients treated at a time when the growth was apparently localized have been high.

Summary

Statistics bearing upon operability and survival of 604 patients in whom the diagnosis of primary carcinoma of the lung was made are presented.

The extension or localization of the lesion and the pathological cell type are the most important factors in survival after resection.

With known and available methods of screening for silent lesions, a significant increase in the salvage rate in primary cancer of the lung should be possible.

In the identical period of the above study (1932–1948), 27 patients were treated by pulmonary resections for tumors diagnosed as bronchial adenoma, infiltrative bronchial adenoma or malignant adenoma. Some authors consider these tumors to be primary carcinomas (adenocarcinoma, Grade I) and include them in their cancer statistics. From a clinical point of view, they should receive special consideration. For example, 26 of 27 patients have been followed recently, and there has been only 1 late death. This patient died of a metastasis that had similar characteristics to the bronchial tumor. One patient has not been followed. Ten of the remaining 25 patients have lived five to fifteen and a half years after operation. In the adenoma series, there were no operative deaths; therefore, since immediate and late results are so totally different in the "so-called" adenoma group, we have not included them in the above paper on true cancer of the lung.

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PULMONARY EMBOLISM*

Analysis of 74 Autopsy Cases Since 1941

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THERE has been no significant decrease in the percentage of cases in which pulmonary emboli were demonstrated at autopsy at this hospital group since the introduction of definitive and prophylactic treatment of venous thrombosis. This therapy includes venous interruption and the administration of heparin and dicumarol, as well as prophylactic exercises, early ambulation and even paravertebral autonomic block in one case. During the thirteen years from 1928 through 1940 pulmonary emboli were found in 7.5 per cent of all autopsies, whereas the figure became 6.7 per cent during the following six-year period (Table 1). The percentage of operations that were followed by pulmonary embolism, as found at post-mortem examination, has decreased somewhat more in the same intervals. Before January, 1941, the figure was 0.13 per cent; since that date it has dropped to 0.09 per cent, but even this decrease is not definitely significant statistically.‡

Because there are so many factors that may change over a period of years, making statistical data questionable, Table 2 was compiled. It shows the various relations between the number of hospital admissions, operations, deaths, autopsies and pulmonary-embolism autopsies per year since 1928. The ratio of operations to hospital admissions has gradually increased over the years. We feel safe in concluding that the other individual ratios have not changed enough to make the above percentages on pulmonary embolism at autopsy lose their significance.

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‡The standard error of the difference for the percentages was calculated according to the formula given in Arkin and Colton.[1] The actual difference between the two figures (7.475 − 6.685 = 0.790) is only 0.22 times the standard error of the difference (3.584). This is definitely not significant, for the chances are more than eight out of ten that the actual difference is an accidental one.

Using the same formula for the percentage of operations followed by embolism as that found at autopsy for the same groups of years, we find that the actual difference between the two (0.0412 per cent) is 2.3 times the standard error of the difference (0.0177 per cent), which may be significant.

The purpose of this study is to attempt to determine why many patients are still dying with pulmonary embolism despite the availability of anticoagulant drugs and the general use of venous interruption in recent years. No analysis of the numerous successfully treated cases was made. Hence this report should not be considered in any sense a condemnation of the methods in use at the present time for the prevention of pulmonary embolism, but rather an indication that the problem is still great even though some of the recent papers lead one to believe that it has been all but completely solved. No attempt has been made to review the literature because this has been excellently done by Neuhof and Klein[2] and Homans.[3]

Seventy-four consecutive autopsy cases with pulmonary embolism in the six-year period from 1941 through 1946 have been studied. Breakdown of so small a number of cases cannot be expected to give statistically significant results, but interesting data can be obtained. Some of these cases have been previously reported in a different type of study by Evans.[4]

There were 41 men and 33 women. Most authors on the subject agree that there is no sex difference, except possibly that the pelvic venous plexuses are more often disturbed surgically in the female, and that this sex group might therefore be more frequently affected.[2, 5] Ages varied from twenty-four to eighty-six years, with an arithmetic mean of sixty years. One might argue that we have not seen a greater decrease in thromboembolic deaths because an older age group has been operated upon in recent years; however, the arithmetic mean of the ages for the first 74 autopsied cases with pulmonary embolism since 1928 was found to be slightly over fifty-eight years. Thus, there is even less than twenty-four months' difference between the average ages of the two groups. It is of interest that Henderson[5] found the average age in 223 patients dying after operation with pulmonary

TABLE 1. *Relations of Pulmonary Embolism at Autopsy to Total Number of Autopsies and Operations.*

PERIOD	NO. OF AUTOPSIES	NO. OF AUTOPSIES WITH PULMONARY EMBOLISM	PERCENTAGE OF AUTOPSIES WITH PULMONARY EMBOLISM	NO. OF OPERATIONS	NO. OF OPERATED CASES WITH PULMONARY EMBOLISM AT AUTOPSY	PERCENTAGE OF OPERATIONS WITH PULMONARY EMBOLISM AT AUTOPSY
1928–1940	2366	177	7.5	88,622	115	0.13
1941–1946	1107	74	6.7	59,894	53	0.09

TABLE 2. *Annual Breakdown of Statistics Concerning Total Number of Deaths, Autopsies, Operations and Pulmonary Embolism.*

YEAR	ADMISSIONS	OPERATIONS	DEATHS	AUTOPSIES	CASES OF PULMONARY EMBOLISM AT AUTOPSY	OPERATED CASES WITH PULMONARY EMBOLISM AT AUTOPSY	RATIO OF OPERATIONS TO ADMISSIONS	PERCENTAGE OF FATAL CASES AUTOPSIED	PERCENTAGE OF AUTOPSIES WITH PULMONARY EMBOLISM	PERCENTAGE OF OPERATIONS FOLLOWED BY PULMONARY EMBOLISM AS FOUND AT AUTOPSY
1928	7,796	4,358	225	181	10	5	0.56	80.0	5.5	0.11
1929	9,601	4,619	266	206	20	13	0.48	77.0	9.7	0.28
1930	9,804	5,554	252	178	15	8	0.57	72.0	8.4	0.14
1931	9,856	5,752	237	154	6	4	0.58	65.0	3.9	0.07
1932	9,137	5,352	231	143	7	5	0.59	61.0	4.9	0.09
1933	9,026	5,523	252	154	10	7	0.61	61.0	6.5	0.13
1934	10,743	6,574	306	201	17	13	0.61	66.0	8.5	0.20
1935	10,578	6,757	305	181	16	6	0.64	59.0	8.8	0.09
1936	12,257	7,804	319	197	23	18	0.64	62.0	11.7	0.23
1937	12,987	8,725	350	199	7	5	0.67	57.0	3.5	0.06
1938	12,988	8,843	389	213	18	13	0.68	55.0	8.5	0.15
1939	13,399	9,128	339	187	16	8	0.68	55.0	8.6	0.09
1940	13,313	9,651	350	172	12	10	0.72	49.0	7.0	0.10
Totals ..	141,485	88,640	3,821	2,366	177	115				
Averages							0.63	62.0	7.47	0.130
1941	13,542	9,355	311	172	10	6	0.69	55.0	5.8	0.06
1942	13,931	9,837	329	172	15	10	0.71	52.0	8.7	0.10
1943	12,956	9,909	358	214	8	6	0.76	60.0	3.7	0.06
1944	12,245	10,368	299	172	10	8	0.85	51.0	5.8	0.08
1945	11,610	9,935	317	200	14	11	0.86	63.0	7.0	0.11
1946	11,932	10,490	315	177	17	12	0.88	56.0	9.0	0.11
Totals ..	76,216	59,894	1,929	1,107	74	53				
Averages							0.79	57.0	6.68	0.089
Grand totals	217,701	148,534	5,750	3,473	251	168				
Averages							0.68	60.4	7.23	0.113

embolism as the primary cause of death to be ten and a third years older than the average age of the patient at operation.

The immediate cause of death in these cases has changed somewhat through the years. In the recent six-year group of 74 cases, embolism was considered the immediate cause in 58. The other causes were as follows: cardiac infarction 3, cardiac failure 3, pulmonary insufficiency 2, pulmonary edema 1, sepsis 2, peritonitis 1, and undetermined 4 cases. It will be noted that only 3 were ascribed to infection. Again, with the first 74 cases since 1928 for comparison (Table 3) pulmonary embolism was considered the immediate cause of death in 48, whereas infection accounted for 11. Because infection is now controllable to a great extent, embolism as the immediate cause of death has become in recent years relatively more rather than less common in this series.

TABLE 3. *Comparison of Recent and Remote Autopsied Cases of Pulmonary Embolism.*

PERIOD	ARITHMETIC MEAN AGE	DEATHS DIRECTLY ATTRIBUTABLE TO EMBOLISM	DEATHS DIRECTLY ATTRIBUTABLE TO INFECTION	PATIENTS OPERATED ON	PATIENTS NOT OPERATED ON
	yr.				
1941–1946 (74 cases) ...	60.0	58	3	53	21
1928–1940 (74 cases) ...	58.1	48	11	50	24

The role of surgery as a causative factor in thromboembolic disease is difficult to evaluate. In the early literature it was especially stressed, but more recently it has been shown that a greater percentage of medical patients than of those subjected to surgery die of pulmonary embolism.[6, 7] In the recent series, 72 per cent (53 patients) underwent operations. This is approximately the percentage of surgical cases in this group of hospitals. It is, however, the group of 21 patients not operated upon that are especially worthy of consideration. Only 5 had mural thrombi in the heart; 2 of these also had leg thrombi. Thus, 16 to 18 of 21 patients who were not operated on died with pulmonary emboli originating from veins rather than from the endocardium. These figures substantiate the views of Hunter et al.[8] that the common denominator of hospitalized medical and surgical patients is confinement to bed irrespective of the type of disease, and that therefore recumbency is probably the greatest single factor in thrombus formation.

The duration of bed rest in this group cannot be determined, for it is not known how long the patients had been in bed before entering the hospital or undergoing surgery. However, the interval from operation to death in patients who underwent a single recent operation is known (Fig. 1). The time extremes are thirty-three hours and thirty-six days, with an arithmetic mean of twelve days. The patient who lived only thirty-three hours postoperatively had no recognized symptoms of thrombosis before the surgical procedure. These time relations are in accord with other data in the literature, which include many more cases and have been reviewed by Neuhof and Klein.[2]

Conclusions regarding the primary sites of the thrombi have been divergent in the literature. Some claim that almost all thrombi arise in the tributaries of the common femoral vein. On the other hand, Moran[9] found that 45 per cent of all male patients who died with pulmonary emboli had prostatic thrombi. In 96 patients who died because of a massive embolus Cohn and Walsh[10] found thrombi in 31 of the femoral or iliac veins, 20 in the pelvic veins (14 periprostatic), 4 in the tributaries of the superior vena cava and 7 in the right side of the heart, and in 37 no thrombus was found. Our figures were similar. In reviewing these figures one must keep in mind that with a changing resident staff in pathology the interest in this problem has waxed and waned so that the primary site of thrombus was searched for with varying degrees of diligence. In Neuhof's[2] series, for example, no primary thrombus was found in over 50 per cent of the cases.

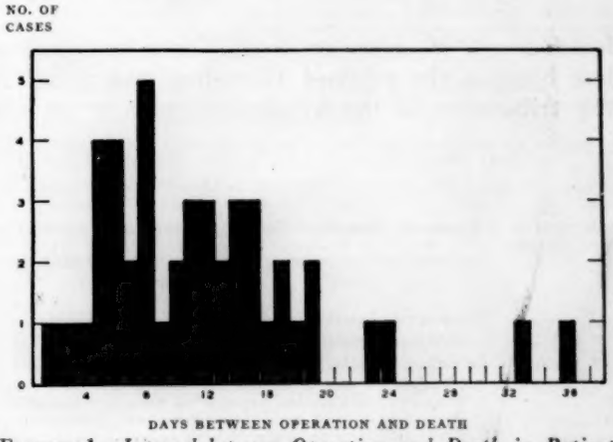

FIGURE 1. *Interval between Operation and Death in Patients Undergoing a Single Operation.*

We classified the primary sites of thrombi into three groups according to the probable value of interruption of the common femoral vein (Fig. 2). The procedure was considered helpful, valueless or questionably helpful (Table 4). The first group

comprises 15 cases in which femoral-vein ligation was considered helpful; in each, a thrombus was found in the veins below the inguinal ligament. The second group consists of 15 cases in which

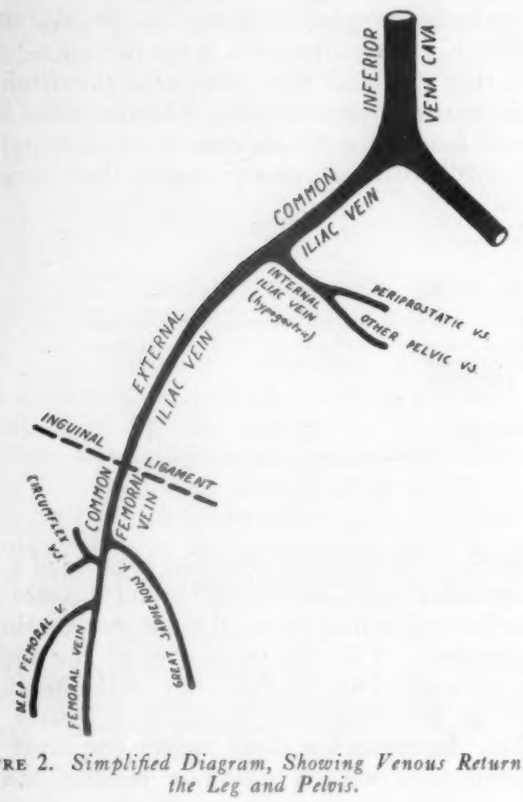

FIGURE 2. *Simplified Diagram, Showing Venous Return from the Leg and Pelvis.*

femoral-vein interruption would have been valueless because the primary thrombus was either in the tributaries of the hypogastric vein or on the cases in which the procedure might have been helpful if it had been performed early enough; it includes the cases in which a thrombus was found in the veins collecting blood from the femoral vein but was proximal to the inguinal ligament—namely, the external iliac, common iliac and inferior vena cava — as well as the 25 cases in which no primary thrombus was found. These figures lead us to conclude that interruption of the common femoral vein is not the ideal approach to the prevention of embolic phenomena.

The greatest problem of all, however, is still the difficulty in recognizing venous thrombosis and benign pulmonary embolism before further embolism results in death. In many of the charts in these 74 cases no evidence of venous thrombosis was given, except possibly a slight rise in pulse and temperature. In other cases, however, the patient had chest symptoms that were diagnosed as pneumonitis, pulmonary edema, atelectasis and coronary thrombosis and remained unrecognized as embolic episodes until death. Evans[4] found in retrospect that 85 per cent of 52 postoperative fatal cases had premonitory signs and symptoms for a sufficiently long time before fatal embolism to have allowed adequate therapy to have been administered.

Roentgenograms are not so helpful as one might think. In only 28 of the 74 fatal cases were there sufficient chest symptoms during the last week of life before the final agonal episode to warrant x-ray examination of the chest. In 18 of these 28 cases pulmonary embolism was not recognized radiographically because of additional coexistent pulmonary and pleural abnormalities as shown by autopsy. These included pulmonary cancer in 4 cases, pulmonary congestion in 3, pneumonitis in 2, and atelectasis, pneumonitis and atelectasis, lung

TABLE 4. *Potential Value of Common-Femoral-Vein Interruption according to Site of Primary Thrombus.*

SITE OF THROMBUS	OPERATED CASES	NONOPERATED CASES	TOTALS
Interruption helpful:			
Common femoral vein or tributaries, or both	12	3	15
Interruption valueless:			
Hypogastric vein or tributaries, or both	5	2	7
Both external iliac and hypogastric veins or tributaries, or both	2	0	2
Right side of heart and inferior-vena-cava tributaries	1	2	3
Right auricle or ventricle, or both	0	3	3
Interruption questionably helpful:			
External iliac vein, with or without its tributaries	1	0	1
Common iliac vein or inferior vena cava, or both	8	3	11
Questionable (leg symptoms without autopsy confirmation)	5	1	6
Questionable (arm symptoms without autopsy confirmation)	1	0	1
Undetermined	18	7	25
Totals	53	21	74

endocardium of the right side of the heart; fatal pulmonary emboli could probably have been prevented in 9 of these by tying off of the inferior vena cava. The third group contains the remaining 44 abscess, chronic empyema, fibrous pleural adhesions and effusion with pneumothorax in 1 each. This leaves only 10 cases with x-ray films available in which pulmonary embolism with or without pul-

monary infarction was the sole pathologic finding in the lungs at autopsy. Chest roentgenograms of 5 of these showed no abnormal densities in the lung fields, so that in all probability the emboli lodged in the pulmonary arteries after the film was taken. In the remaining 5 cases, the greatest misinterpretation of shadows cast by lung infarcts as the only pathologic process found at autopsy was the diagnosis of pneumonitis or bronchopneumonia (4 cases).

Since 1941 the clinicians at this group of hospitals have been actively treating patients to prevent pulmonary emboli. The Lahey Clinic alone treated 184 postoperative cases of venous thrombosis with or without pulmonary embolism from 1942 through 1946. In these only 3 patients died of thromboembolic disease.[11] Yet the most surprising result of our study was that in the 74 autopsied cases only 5 patients had received any definitive treatment to prevent embolism. This emphasizes the difficulty in recognizing venous thrombosis and benign pulmonary embolism. Three of these patients died with embolism as the immediate cause of death. The other 2 died of unrelated conditions, and the minor emboli were incidental findings.

Thus, despite the many enthusiastic reports on venous interruption and anticoagulant therapy for the prevention of pulmonary embolism, the problem is still far from solved, and the careful search for the slightest symptoms suggestive of venous thrombosis and pulmonary embolism remains a most important responsibility of every physician.

Summary

The incidence of autopsied cases with demonstrable pulmonary embolism has dropped only from 7.5 to 6.7 per cent since the use of definitive and prophylactic treatment of venous thrombosis, including venous interruption and the administration of heparin and dicumarol.

The decrease is slightly greater when the percentage of deaths coming to autopsy with pulmonary emboli is compared to the number of operations performed, rather than to the total number of autopsies done. From 1928 through 1940 it was 0.13 per cent, whereas from 1941 through 1946 it was 0.09 per cent. From a statistical standpoint this might be a significant decrease.

A study of the 74 cases autopsied from 1941 through 1946 was undertaken to help explain this relatively insignificant change. The cases were analyzed according to age, sex, immediate cause of death, primary site of thrombus, medical versus surgical status and preventive therapy.

In only 5 of the 74 autopsied cases of pulmonary emboli did the patients receive specific therapy for the prevention of embolism. This suggests that the primary problem is the difficulty in the diagnosis of venous thrombosis and nonfatal pulmonary embolism.

Despite the availability of various means of preventing pulmonary embolism, the condition remains a great problem.

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SERUM POTASSIUM LEVELS IN DIABETIC COMA*

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THE clinical importance of variations in the serum potassium level during treatment of diabetic coma has been emphasized by the reports of Holler,[1] Martin and Wertman,[2] Nicholson and Branning,[3] Frenkel, Groen and Willebrands[4] and Nadler, Bellet and Lanning.[5] They reported low potassium values in certain patients undergoing treatment for diabetic coma, with fatal results unless suitable potassium solutions were administered promptly. On the other hand, studies of serum potassium levels in conditions of shock, cardiac or renal failure indicate that if potassium is given as a therapeutic measure to patients with impending or actual renal failure, a dangerously high level of potassium in the blood serum may also have a fatal result. Today, the physician responsible for the treatment of patients in diabetic coma, must have constantly in mind the possibility that critical alterations in the level of blood serum potassium may result in a fatal outcome unless proper treatment is given in an early stage.

The causes of a decline in the serum level of potassium from a normal of around 5.0 milliequiv. per liter to levels of 3.0 milliequiv. or less are varied. It has long been known that during acidosis, particularly of the diabetic type, there is an immediate and increasing loss of potassium in the urine. This potassium comes chiefly from the cells through disturbance in cellular permeability occurring during diabetic coma. A considerable loss of potassium in the urine may occur before extracellular levels are seriously affected. In cases of diabetic coma, such a loss may be very severe. This is especially true in previously undiagnosed cases.

It is the purpose of this paper to present 6 cases of diabetic coma in which routine potassium observations were made. All cases were studied in the George F. Baker Clinic of the New England Deaconess Hospital, and are reported to show the effect of treatment without glucose on varying degrees of acidosis.

CASE REPORTS

CASE 1. This patient, a child, was not known to have diabetes until she was found in a drowsy state after several days of nausea and vomiting. On admission to the hospital the blood sugar was 400 mg. per 100 cc., and the serum potassium was 3.1 milliequiv. per liter. At the end of 17 hours of treatment the serum potassium fell to 2.5 milliequiv., but she had no referable symptoms and made an uncomplicated recovery with only 66 units of insulin and 1000 cc. of physiologic saline solution intravenously (Table 1).

TABLE 1. *Serum Potassium Levels in Diabetic Acidosis.*

Case No.	Blood Sugar	Plasma Carbon Dioxide	Serum Potassium		Duration of Treatment	Insulin Dosage	Twenty-Four-Hour Parenteral Injection of Physiologic Saline Solution	Clinical State
			on admission	lowest				
	mg./100 cc.	*milliequiv./liter*	*milliequiv./liter*	*milliequiv./liter*	*hr.*	*units*	*cc.*	
1	400	9	3.10	2.50	17	66	1,000	Patient drowsy
2	463	6	5.80	3.80	24	208	3,000	Patient semicomatose and anuric
3	450	5	3.75	3.12	20	270	2,500	Patient drowsy
4	805	9	4.43	3.60	20	450	3,000	Patient drowsy
5	296	10	4.30	4.10	10	165	2,000	Patient conscious
6*	765	10	5.40	3.30	4	340	4,000	Patient in coma, with shock and anuria, followed by death

*Included through the courtesy of Dr. Robert Brownlee.

CASE 2. A 19-year-old girl in diabetic acidosis for the second time had made very little attempt to control her diabetes. She had not followed the prescribed diet, nor had she checked her urine for sugar. Polyuria and polydipsia had been present for several weeks. Three days prior to admission abdominal pain had developed, followed in 48 hours by nausea and vomiting. After twenty-four hours of vomiting she was admitted to the hospital in a semiconscious state. The first hour's treatment consisted of intravenous administration of physiologic saline solution and 200 units of regular insulin. The admission potassium of 5.8 milliequiv. per liter fell to 3.8 milliequiv. in the next 24 hours. Recovery was uncomplicated.

CASE 3. This young woman followed very closely the pattern of Case 2, except that vomiting was present 48 hours prior to admission. The serum potassium levels were correspondingly lower, but her recovery was uneventful.

CASE 4. A middle-aged man had not been seen in the clinic for 2 years and had made very little effort to control his diabetes. In spite of the distressing symptoms of uncontrolled diabetes, he had continued eating a diet liberal in carbohydrate and maintained an inadequate insulin dosage. Nausea and vomiting began while he was on his way to the hospital from another state. After 4½ hours of vomiting he arrived at the hospital in fairly good condition, with a blood sugar of 805 mg. per 100 cc., and a carbon dioxide content

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of 9 millimols per liter and a serum potassium of 4.43 milliequiv. per liter. Treatment, which was immediately instituted, consisted of 3 liters of physiologic saline solution and 450 units of regular insulin. The lowest serum potassium, which was determined 20 hours after admission, was 3.6 milliequiv. per liter.

Case 5. A young adult in mild acidosis had a carbon dioxide content of 10 millimols per liter and a blood sugar of 296 mg. per 100 cc. This patient required only 165 units of regular insulin, and the serum potassium fell from a normal of 4.3 to only 4.1 milliequiv. per liter.

Case 6. An elderly woman with known diabetes who had never required insulin was found comatose in her apartment after approximately 24 hours of acidosis. After hospitalization and institution of treatment she responded to 340 units of insulin and roused from her coma. She continued anuric, however, and suddenly vomited and aspirated a large amount of material in spite of previous gastric lavage. Death was due to asphyxia.

Discussion

The cause of low serum potassium levels may be contrasted with those of high values, which occur almost without exception in patients with oliguria or anuria. Cases 2 and 6 in this series illustrate this point well. Each patient was anuric for a variable period before treatment, and in each, the admission potassium level was slightly above normal limits. As early as 1915, Smillie[6] described potassium poisoning in nephritis when potassium salts were administered therapeutically. Van Slyke[7] points out that a fall in systolic blood pressure of 40 to 60 may in itself be sufficient to cause anuria. Such levels are not at all rare in severe diabetic coma. It should be recalled, however, that elevated serum potassium levels in diabetic acidosis and shock do not necessarily reflect the true state of the tissue cells of the body. Prior to the late stage of anuria and hemoconcentration, there is usually a prolonged period of diuresis with resultant potassium loss. Thus, the ambiguous condition results in which there is a total body deficit of potassium in the presence of an elevated serum potassium level. This condition must be borne in mind during treatment, and a false sense of security avoided when one finds a normal or slightly elevated level on admission. The period of early recovery from coma may indeed be one of the most dangerous periods from a chemical standpoint. Thus, the serum potassium levels in Cases 2 and 6, which were slightly elevated on admission, fell to below the normal range in a period of four to twenty-four hours after recovery from coma. This factor makes imperative the early feeding by mouth of orange juice and other foods rich in potassium.

Various aspects of emergency serum potassium levels in diabetic coma are presented in Table 2. The diagnosis of low serum potassium is particularly to be borne in mind when a patient in diabetic coma has received glucose solution by vein early, and, at a period varying from four to twenty-four hours after the beginning of treatment, shows the characteristic clinical signs. So far, no patient in this series (or in other reports from the literature) who has not received considerable amounts of glucose during the first few hours of treatment has shown seriously low serum potassium levels. The actual diagnosis is, of course, best established by serial analyses of the serum. Since this determination is not always obtainable, however, one should always remember that (in patients with low serum potassium levels) the sudden development of marked muscular weakness or actual flaccid paralysis may mean hypopotassemia. In 1 or 2 patients a peculiar fish-mouth facial expression has been noted. An important diagnostic aid is afforded by the electrocardiogram, in which the changes accompanying low serum potassium values are in marked contrast to those observed when the serum potassium value is abnormally high. Thus, in patients with a low value, low-voltage QRS complexes and lowering or actual inversion of the T waves are observed. The ST segments may become depressed, and the QRS interval may be prolonged. In contrast, the electrocardiogram in patients with a high level may show an absence of the P waves, wide QRS complexes and high, peaked T waves.

Table 2. *Emergency Serum Potassium Levels in Diabetic Coma.*

Potassium Level	Causes	Diagnostic Features	Treatment	Prevention
Low	Insufficient or delayed treatment of acidosis; extreme diuresis, with loss of potassium in urine; excessive administration of glucose, with glycogen deposition; vomiting.	Sudden flaccid paralysis progressing to respiratory paralysis 10-24 hr. after treatment with glucose and insulin; fish-mouth facial expression; low-voltage QRS complexes and flat or inverted T waves; fall in serum potassium from normal of 4.1 to 5.6 milliequiv. per liter to less than 3.0 milliequiv.	Potassium phosphate (2 gm.) with potassium dihydrogen phosphate (0.4 gm.) intravenously; 100 cc. of 2 per cent potassium chloride solution intravenously.	Withholding of glucose from patients in coma; rapid treatment to shorten acidosis; early feeding of potassium-containing food (oatmeal, orange juice and so forth).
High	Oliguria or anuria; administration of potassium in acute or chronic renal failure.	Paralysis (flaccid) or spastic type; absent P waves, wide QRS complexes and high, peaked T waves; high value for serum potassium (levels exceeding 12 milliequiv. per liter may be fatal).	Cause should be treated; administration of glucose and insulin.	No administration of potassium (without sure indication)

A serum potassium value exceeding 12 milliequiv. per liter may be and has been reported as fatal. Paralysis may be flaccid or of the spastic type. A more detailed discussion of electrocardiographic changes in various conditions is beyond the scope of this paper. The subject is adequately covered by Milhorat[8] and by Brown, Currens and Marchand[9] and others.[5, 10, 11]

Currens and White[12] noted a fall in the serum potassium levels during treatment of patients with Addison's disease by means of desoxycorticosterone acetate. They believed that the weakness associated with this treatment was often due to potassium loss. The changes in the electrocardiogram accompanying high values of serum potassium are discussed by Tarail[13] in a case of uremia and have also been described by Thorn,[14] who cites the article on the clinical syndrome of potassium intoxication by Finch, Sawyer and Flynn.[15]

Treatment of low serum potassium has proved successful in the cases of Holler,[1] Nicholson[3] and Frenkel,[4] who use potassium chloride orally or begin with 100 cc. of a 2 per cent solution intravenously. Because glucose requires phosphorylation before it can be utilized, it seems wise to employ a combination of potassium and phosphate in cases that require intravenous administration of potassium salts. In all such cases, however, the injection should be given slowly to avoid a transitory rise in the serum level to a dangerous concentration, and the diagnosis should be established with certainty either by means of actual serum analysis or unequivocal symptomatology and electrocardiographic findings. For high serum potassium values, treatment of the underlying cause of renal insufficiency is indicated.

The prevention of low serum potassium levels is possible by early and more rapid treatment of diabetic acidosis. Every effort should be made to shorten the period of serious diabetic acidosis. This means prompt use of insulin, when possible, long before the patient becomes drowsy. Since, thus far, no patient has developed serious clinical hypopotassemia unless intravenous glucose solution was administered during the first six to eight hours of treatment, one of the fundamental principles is to give no glucose in the first hours of treatment in diabetic acidosis.

The excessive administration of glucose, especially with insulin, results in a rapid deposition of glycogen in the liver, associated with an intracellular shift of potassium, as shown by the experimental work of Fenn.[16,17] Weissberg's[18] patient received glucose intravenously and subsequently developed a serum potassium level of 1 milliequiv. per liter — the lowest level recorded in the literature. In the case reported by Tuynman and Wilhelm[19] a low serum potassium level with respiratory difficulty developed after the patient had received glucose from the onset of treatment of diabetic coma. They cite the work of Allott and McArdle[20] in which 3 cases of periodic familial paralysis showed that attacks of paralysis, clinically identical with a naturally occurring attack, could be produced by the administration of glucose by mouth and by insulin but more consistently by glucose and insulin together.

At the New England Deaconess Hospital during the three years ending April 1, 1948, 91 consecutive cases of diabetic coma have been treated without glucose and without a single death in spite of such complications as pneumonia, cerebrovascular accidents, heart disease, coronary occlusion and chronic nephritis. The mere storage of excess glycogen has no value during the first few hours of the emergency in diabetic coma. If 200 gm. of glucose is given in the first few hours of treatment of diabetic acidosis, no more than 10 gm. of glucose can be oxidized each hour.[21] The remainder must be excreted in the urine, deposited as glycogen (thereby producing a further reduction of serum potassium) or converted into fatty acids, as shown clearly by the isotopic studies of Stetten.[22]

Diabetic coma should always be considered an emergency in which the main object is to give an adequate amount of insulin in the first hours, to restore fluid balance promptly and to guard against potassium abnormalities. In most cases, it is possible to start giving food orally after six to twelve hours. It is the practice of this clinic to use food, such as oatmeal gruel and orange juice, which contain potassium, as soon as the stomach will retain it. It is therefore timely and important in all cases of coma to wash out the dilated stomach and give an enema, to prepare the stomach for the taking of food at as early a period as possible. Naturally, in patients remaining unconscious for twenty-four to forty-eight hours this procedure cannot be carried out, and intravenous feedings utilizing glucose become necessary. In these cases the danger that hypopotassemia will develop must be kept constantly in mind. The prevention of fatal potassium intoxication may depend chiefly upon the avoidance of errors and the omission of potassium without definite indications.

Summary

Six cases of low potassium levels in diabetes from the George F. Baker Clinic of the New England Deaconess Hospital are reported and discussed.

Emergency serum potassium levels may occur during the treatment of diabetic coma and must constantly be kept in mind.

The cause, diagnosis, treatment and prevention of high and low serum potassium levels are compared.

For the prevention of dangerously low serum potassium values in diabetic coma, the omission of glucose administration during the first hours of treatment and more aggressive use of insulin are recommended.

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DELAYED PNEUMONIA AND URTICARIA FOLLOWING BRONCHOGRAPHY

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PNEUMONIA complicating bronchography has been described.[1-4] Some of the cases previously reported have been attributed to infectious factors, owing to the carrying down of infecting organisms from the upper respiratory tract. Waldbott[4] has emphasized the role of iodized oil in producing obstruction of the small bronchi followed by atelectasis of lung tissue and pneumonitis or massive collapse of a lung when a main bronchus is involved. Other authors have reported cases of pneumonia believed to be due to an allergic reaction to the iodide component of lipiodol.[2] All the reported cases, whether infectious or allergic, were characterized by a rapid onset of pneumonia, which usually occurred within twenty-four or forty-eight hours.

A number of fatal cases have also been reported. In 1 of these cases death occurred about seventy-five minutes after instillation of the iodized oil.[4] On post-mortem examination, both lungs were atelectatic, and the tracheobronchial tree was completely filled with thick, extremely tenacious mucus, which was found plugging even the smallest bronchioles. These deaths were considered to be allergic and were directly attributed to the iodine fraction of the iodized oil used.

More recently, 3 cases of pneumonia following the use of lipiodol in asthmatic patients were described that differed from those previously reported.[5] A delayed onset of pneumonia was noted, varying between nine and twelve days. In addition, these cases were characterized by certain allergic manifestations such as urticaria, blood eosinophilia and positive sensitivity tests with iodide-containing compounds. The delayed pneumonic reaction was described as resembling the delayed reaction of serum sickness. In view of the increasing use of lipiodol as a diagnostic procedure in the field of thoracic disease, one may expect to see more cases of this type of complication following bronchography.

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Case Report

W. P., a 59-year-old man, was admitted to the Municipal Sanatorium on July 17, 1947. There was no familial history of tuberculosis. He stated that in November, 1940, he had suffered an attack of "pleurisy and pneumonia" for which he had been treated at home. A chest x-ray film taken in 1941 was reported as normal. He subsequently became an employee of the Municipal Sanatorium, where a chest x-ray examination in June, 1946, showed a healed primary complex, with calcifications at both hilar areas. In July, 1947, a nonproductive cough developed, and a chest film revealed an area of infiltration in the left lung. He was admitted to the Sanatorium as a suspected case of pulmonary tuberculosis.

Physical examination was within normal limits except for a soft blowing systolic murmur transmitted to the left. The lungs were clear. The blood pressure was 160/90. The sedimentation rate was 8 mm. per hour (Westergren method). A Mazzini test was negative. The urine showed no abnormal findings. During the period of hospital observation, ten concentrated sputum smears and four gastric cultures were negative for tubercle bacilli. X-ray examination of the chest revealed a mottled infiltration in the left-upper-lung field containing a central, oval-shaped area of homogeneous opacity. This infiltration showed some increase in its extent shortly after admission, but subsequently regressed.

Bronchoscopic examination performed on April 12, 1948, was negative; however, the left-upper-lobe bronchus could not be visualized. It was believed that a bronchogram should be taken because of the possibility of an underlying bronchiectatic condition. Accordingly, a bronchogram was taken

on the left side on July 31 (Fig. 1). About 8 cc. of lipiodol was instilled into both lobes of the left lung by means of an intratracheal catheter. No immediate untoward reaction was noted. The bronchogram showed suggestive bronchiectatic changes in the left-upper-lung field.

Fourteen days later the patient first noted the onset of productive cough. The temperature, which had been normal, rose to 100.4°F. At the same time, an extensive itching skin

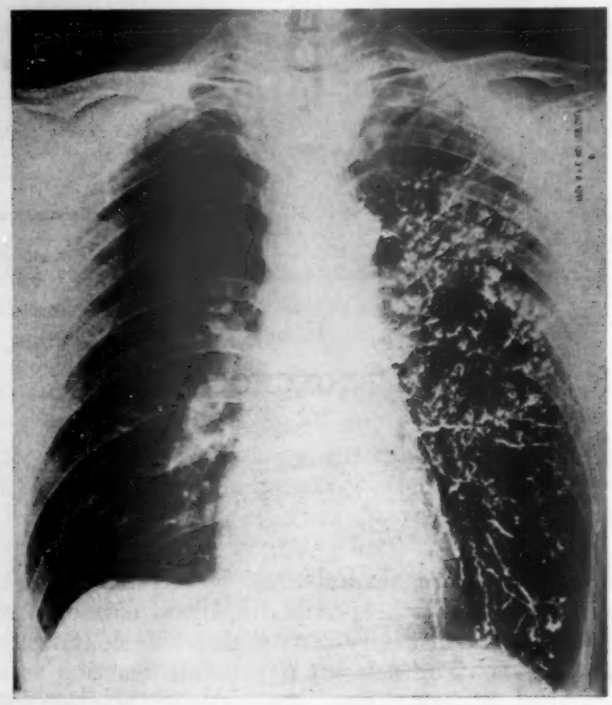

FIGURE 1. *Bronchogram of the Left Lung, Taken on July 31, 1948.*

Note the suggestive bronchiectatic changes in the left-upper-lung field.

eruption, which was urticarial and covered the arms, legs and trunk, developed. On questioning, the patient stated that he had had a similar eruption, which he described as "hives," on several previous occasions.

A chest x-ray film taken on August 18 (Fig. 2) showed an extensive area of pneumonia occupying the lower-left-lung field. Numerous small densities representing retained lipiodol were visible throughout the left lung. Rales were heard at the left base for the first time. In spite of the rather extensive area of pneumonia visible on x-ray study, the patient appeared surprisingly comfortable. The temperature fell by lysis and reached normal levels after 5 days. A chest x-ray film taken on September 3 (Fig. 3) showed some regression of the area of pneumonic density, which still occupied the base of the lung. No allergic studies were performed at the time of the acute illness. However, a blood count done 2 weeks after the disappearance of the urticarial eruption did not reveal any increase in eosinophilic leukocytes. Iodine-sensitivity tests were also carried out at this time. An oral test with potassium iodide, a scratch test with lipiodol and an intracutaneous test with diodrast were negative.

DISCUSSION

Immediate allergic reactions to iodine in hypersensitive persons have been described.[6] These reactions are analogous to those produced by serum therapy in patients sensitive to horse serum. In nonsensitized subjects a form of induced sensitivity that is characterized by a definite incubation period of one to three weeks may occur; the familiar phenomenon of serum sickness belongs in this category. Urticarial reaction of a serum-sickness type occurring after a definite incubation period is a well known complication of penicillin therapy. As described by Kolodny and Denhoff,[7] this reaction is a delayed type because of the time needed for development of the newly induced sensitivity.

A similar time lag occurs in the type of pulmonary reaction to lipiodol in the case described above. The interval before symptoms appear may be from seven to fourteen days and even longer. The symptoms may be acute and may resemble the onset of a bacterial or virus pneumonia. Urticaria is frequently seen. Severe rashes of urticarial type occurring seven to ten days after injection of iodized oil have been described by Scadding.[8] Cough, expectoration of a mucoid spu-

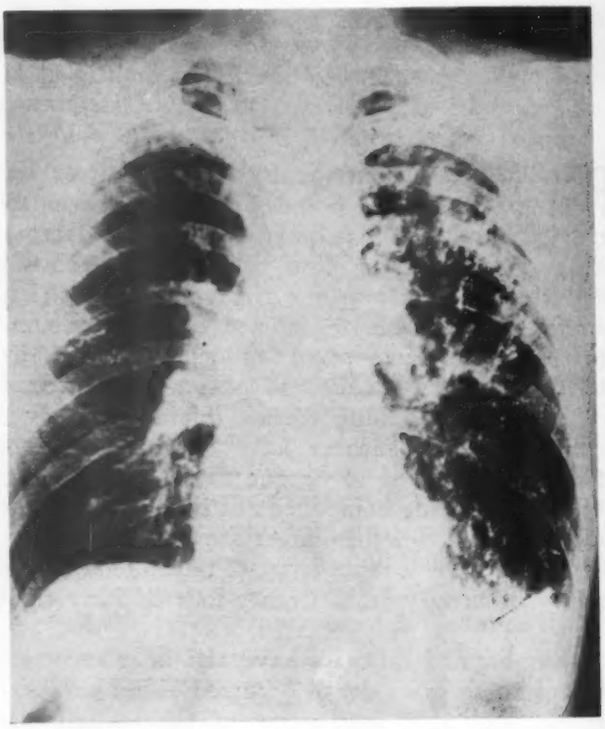

FIGURE 2. *Film Taken on August 19, 1948, Showing Extensive Area of Confluent Pneumonia at the Left Base.*

There are numerous small area densities representing retained lipiodol in the remainder of the left lung.

tum and fever are prominent symptoms. The temperature is often low grade, whereas the extent of pulmonary involvement may be quite massive. Rales are heard over the involved portion of the lung field, usually at the base. Roentgenographic examination often reveals a confluent type of pneumonic density conforming to the area of distribution of lipiodol. Treatment is symptomatic. In the

cases of lipiodol pneumonia originally reported[5] sulfathiazole was employed but later discarded since no evident shortening of the period of illness was observed. It is possible that antihistaminic drugs exert a favorable influence on this type of pulmonary infiltration, and it is suggested that this form of therapy be employed.

A history of either familial or personal allergy is usually obtained in these cases. In such an event, iodine-sensitivity tests should be performed. The intracutaneous test with diodrast is perhaps as valuable as any.[9] However, a negative skin test prior to bronchography is no safeguard against the development of a delayed lipiodol reaction.

In the case reported above, it should be noted that iodine-sensitivity tests were negative about two weeks after the disappearance of the urticarial eruption. Peck and his co-workers[10] have described a similar finding in cases of urticarial reaction due to penicillin. They believe that although a positive reaction to a penicillin test is helpful in confirming a diagnosis of this type of allergy, a negative reaction does not exclude it. Since the reaction to penicillin may be positive in more than a third of such persons, the test is considered worth doing in an effort to prove that an eruption is due to penicillin. Furthermore, it has been pointed out by Lyons[11] that the induced urticarial form of penicillin allergy is often temporary, even transient. This observation has been borne out by Peck et al., who describe 2 patients whose initial reactions to the cutaneous test were positive, but in whom retests gave negative results. They imply that the mechanism by which the urticarial eruption is terminated is related to the reduction in sensitivity that may follow a violent reaction. It is possible that the status of the iodine-sensitivity test in cases of delayed lipiodol reactions is analogous to that obtaining in cases of delayed penicillin reactions.

In view of the foregoing observations, it is evident that an effort should be made to keep patients under observation for at least two weeks after bronchography. This is not always the case with clinic patients, who are frequently discharged after negative bronchographic tests. Iodine-sensitivity tests should be performed in patients who give a familial or personal history of allergy. In the presence of a positive test, bronchography might still be performed if the information to be gained therefrom is considered necessary for the carrying out of a surgical procedure such as pulmonary resection for localized bronchiectasis. In such cases an effort should be made to avoid overfilling of the bronchial tree. Trapping of lipiodol in the alveoli by explosive coughing should also be avoided. This can be accomplished by emptying of the bronchi, with postural drainage after completion of the examination. In addition, the use of antihistaminic drugs as a prophylactic measure against allergic reactions for at least two weeks after bronchography is worthy of consideration.

Summary

A case of delayed pneumonia and urticaria following bronchography in an allergic person is described.

The mechanism of delayed reactions to iodized oil is discussed.

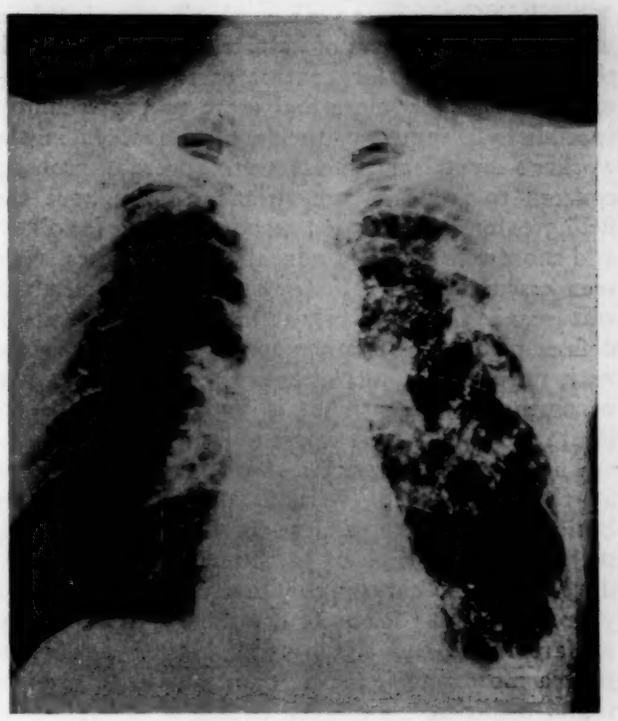

FIGURE 3. *X-ray Film Taken on September 3, 1948. Note the decrease in the extent of the pneumonia in the left lung.*

The importance of keeping patients under observation for two weeks after bronchography is emphasized.

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MEDICAL PROGRESS

INDUSTRIAL MEDICINE

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DURING the past year emphasis was placed on the social phases of industrial medicine. With the responsibility for control of the physical environment falling more and more to the technical specialists in industry, the factors involved in the worker's mental and social welfare were of growing concern to industrial physicians. Throughout the field greater co-ordination among all those involved, and more research and educational activities, were seen essential. As industrial medicine reaches full stature, the further necessity for it to assume a well defined place in the complex social scheme, consistent with the interests of the entire medical profession, becomes apparent. What is to be its scope — its limitations? The question is still unanswered.

The industrial physician today may be said to be at the crossroads.[1] Without deviating from the basic tenets of the profession, he has been compelled to adapt himself to the demands of a fast-developing and changing industrial economy. Each year the various forms of health insurance, group-welfare funds and sick-benefit and pension plans play a more significant part in relation to occupational medicine. The very nature of the industrial practitioner's work, dealing as it does with a specified segment of society, has placed him in an anomalous position. For example, although he adheres in theory to the established precept of the patient's free choice of a doctor, he cannot but violate it in actual practice.

Only further exploration of the situation — from the standpoints of industry, the medical profession and the community in general — can determine how far and how fast industrial medicine will travel. "The ultimate possibilities may be considered as narrowly or as broadly as vision and faith will permit."[2]

The continuing introduction of new manufacturing materials and processes brought scientific advances over a wide front, but no startling discoveries were announced. Many of the investigations into potential health hazards in various industries were incomplete. However, such important developments as were noted are reviewed below.

BERYLLIUM AND ATOMIC RADIATION

The epidemiology of beryllium poisoning is not completely understood, but the various clinical manifestations produced by this metal have been more clearly defined and definite progress has been made toward its control.[3-5] There is no longer any controversy about its toxicity since a significant health hazard can be shown to exist wherever beryllium dust or fume contaminates the atmosphere.

The contact dermatitis that appears in from three to ten days in workers processing beryl ore is of relatively minor consequence, but may be a precursor to the development of the acute or chronic form in susceptible persons. Approximately 50 per cent of workers will develop skin manifestations from contact with beryllium compounds that contain an acid radical, notably beryllium sulfate or beryllium fluoride.[6] Ulceration may occur if a crystal of a soluble beryllium salt is introduced into the skin. These ulcerations do not heal readily until the base is curetted. Granulomatous skin lesions similar to sarcoid occur occasionally in people with the chronic form of pulmonary disease and are considered part of the generalized process. Grier et al.[7] and Shook and Powell[8] have recently described subcutaneous granulomas at the site of lacerations in people who have suffered cuts from fluorescent lamps coated with a zinc manganese beryllium silicate. The pathology of these granulomas is similar to that found in the lungs in cases of chronic pulmonary granulomatosis. Treatment by complete excision is effective. Disposal of burned-out fluorescent tubes is a hazard. Such tubes should be broken under water or disposed of intact, so as to prevent liberation of dust and possible implantation under the skin.

Exposure to soluble salts may produce chemical inflammatory changes in the respiratory tract and conjunctiva. Nasopharyngitis and tracheobronchitis are usually of transient nature and clear up upon removal from exposure. Acute pneumonitis is more serious and not infrequently fatal. The incidence and severity are related to the degree of exposure, the nature of the beryllium compound, particle size, surface area and so forth and to individual factors of susceptibility. The characteristic symptoms are dyspnea, cough, chest pain and cyanosis. Pulmonary infiltration undistinguishable by x-ray examination from generalized bronchopneumonia develops within a period of one to three weeks. Recovery, or death in the fulminating type, occurs within days to several months. At autopsy

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the lungs show a diffuse bronchopneumonitis similar to that in any chemical pneumonitis. The liver and spleen are enlarged and show changes consistent with an overwhelming chemical toxemia. The treatment is supportive and consists chiefly of oxygen, bed rest, antihistaminics and penicillin as a prophylactic.

These acute cases have occurred among workers engaged in the refining of beryl ore, in the manufacture of fluorescent lamps, in ceramics and in the machining of the pure metal. The use of beryllium copper products is not harmful, but when these alloys are ground, melted or tooled, contamination of the atmosphere with dust or fume may occur and produce some form of the disease. The highest incidence of acute pneumonitis is found in exposure to beryllium sulfate or fluoride, and cases have been known to develop after a single twenty-minute exposure to high concentrations.[9] Maximum allowable concentrations have not been agreed upon but are judged to be in the range of 25 microgm. per cubic meter for the acute disease. Engineering revisions of the industrial processes have reduced the incidence of acute pneumonitis to a very low level. Medical control by weekly examination, with particular emphasis on the vital capacity, which is diminished early, and prophylactic lay-off often lessen the severity and limit the tracheobronchial form from progressing.

The chronic form of the disease is characterized by a long delay between exposure and the onset of symptoms. Anywhere from four months to six years after the cessation of exposure, shortness of breath and loss of weight may develop. These symptoms continue until the patient dies, although some seem to have made a partial recovery. Pascucci,[10] in a comprehensive review of the roentgen findings describes two types: a bilateral, uniformly widespread, diffuse granulomatous lesion and nodular infiltration. This fine, disseminated, granular type of infiltration is characteristic and not produced by any other definite pathologic entity. Pulmonary changes visible on x-ray study may exist without significant or any symptomatology. No clear-cut correlation can be drawn between the clinical features of the disease and the roentgenologic appearance of the lesions. Autopsy in the chronic cases shows a diffuse, nodular, granular, inflammatory response characterized by giant cells, epithelioid cells, plasma cells and inclusion bodies and by fibrosis. Pulmonary hypertension from reduction in the size of the pulmonary vascular bed is the most common cause of death. Other viscera, such as the hilar lymph nodes and the liver and spleen, show granulomatous infiltration and extensive fibrosis similar to that in the lung.

The vast majority of patients with a chronic disease give no history of an acute episode. Cases of the chronic disease have also occurred in people who were never exposed within an industrial plant but lived in close proximity to a factory using a beryllium compound, as well as in the families of workers, the exposure apparently produced indirectly by contamination with work clothes. Follow-up examination on a number of workers with the acute disease indicates that most of them have recovered completely. But there are several cases that after a delay of months to years developed the typical clinical picture of the chronic disease. The presence, however, of such cases and the known long delay in onset leaves the question of progress from acute to chronic still undecided.[11]

The prevention of the chronic case is difficult, but periodic examinations of exposed people, including x-ray films of the chest and vital-capacity determinations, are necessary for early diagnosis. Plant sanitation, as well as obligatory washing, showers and change of clothing, is essential for control. Substitution or segregation of beryllium processes will limit the incidence of the chronic disease.

Significant progress has been made in the determination of beryllium in air and body tissues.[12] Analyses of normal tissue indicate that the amount of beryllium is less than 0.05 microgm. per 100 gm. of wet-lung tissue and that normal urine contains no beryllium. Acute cases may show as much as 180 microgm. per 100 gm. of lung. The urine of exposed workers who show no symptoms may contain as much as 1.2 microgm. of beryllium per liter, whereas those suffering from acute pneumonitis may show as much as 4.2 microgm. of beryllium per liter of urine. The urine of patients with chronic granulomatosis varies from negative to 1.4 microgm. of beryllium per liter. Lung tissue in chronic cases contains between 0.05 and 1.6 microgm. The variability and the technical difficulties of doing the beryllium determination in tissue indicate that reliance should not be placed on beryllium tissue content as pathognomonic of the disease, but rather as evidence of exposure to beryllium.

Industrial development of atomic energy has not progressed to the point where it is of significance to most industrial physicians. Results of experimentations, however, by workers associated with the atomic program have appeared in quantity in the literature. Of particular interest to the industrial physician is the evidence that hematologic studies for the reduction of lymphocytes in the peripheral blood are still the most sensitive indicator of exposure to ionizing radiation.[13] More caution in exposure to radiation is beginning to be prevalent among health physicists, who suggest that the tolerance dose be decreased from 0.1 r to 0.05 r per day, or a maximum of 0.3 r per week.[1]

Pneumoconiosis

Although a large number of articles were published describing the occurrence, distribution and characteristics of silicosis in various dusty indus-

tries, no significant revisions were suggested in the diagnosis, epidemiology, management or control of the disease. It is interesting in this connection to note the finding of McCormick et al.[15] that in the processing of leaf tobacco the inhalation of hygienically significant free silica in the tobacco dust may present a hazard.

Exposure factors were more carefully studied in several articles, with special reference to particle-size distribution. Hatch and Kindsvatter[16] indicate that particles below 0.5 microns may be of greater import than was previously thought. The importance of quantitative evaluation of the influence of particle size upon toxic activity of dust is stressed,[17] and Holden and his associates[18] state that the free silica content of the fraction below 5 microns provides a more significant measure of the silicosis dust potentiality than the total dust analysis.

More cases of bagassosis, a pulmonary disease resulting from the inhalation of bagasse dust, were reported.[19] Bagasse, a sugar-cane waste product, is used in the manufacture of insulating and acoustic board, and its dust produces sudden cough, severe dyspnea, chills and fever and occasional hemoptysis. X-ray study discloses diffuse mottling of both lungs, which clears in from two weeks to several months. Numerous etiologies have been suggested,[20] but the most recent work of Schneiter et al.[21] indicates that the cause of the disease is *Aerobacter cloacae* (a soil bacterium also present in cotton) acting in conjunction with the inhaled particles of the irritant bagasse dust.

Aluminum and some of its oxides have been shown experimentally to inhibit the action of silica in producing silicosis, and extensive prophylactic and therapeutic clinical trials are presently under way. A new apparatus for dispensing the aluminum hydroxide dust is described by Church and Ingram.[22] Riddell[23] cautions, however, against the acceptance that the treatment is of value since no conclusive objective evidence has as yet been brought out. The effect of aluminum inhalation in human beings is apparently chiefly psychologic in its effect. Although most of the evidence indicates that alumina is harmless, the finding of pulmonary fibrosis and pneumothorax in workers exposed to bauxite in the manufacture of corundum has been reported.[24] The fume generated in this process consists of finely divided silica and amorphous alumina. Similar experiences from Germany and Sweden[25] corroborate that some processes involving the manufacture of aluminum compounds produce a form of pneumoconiosis. A field study on the effect of alumina and silica is provided in the pottery industry, in which workers are exposed to finely powdered alumina and flint.[26] This group of workmen, although daily exposed to finely powdered alumina in greater intensity and over a longer period than existing methods of treatment could cope with, showed not only new cases of silicosis but also known cases of the advanced form. Baetjer,[27] in a study of the effect of Portland-cement dust reported no effect on animal resistance to lobar pneumonia, differing in this respect from the effects of quartz and feldspar dust.

Specific Chemicals

The toxicologic properties of a host of new industrial chemicals were reported during the past year. Revisions of certain phases of knowledge of some of the older ones were also made. The absorption of lead through the skin was found to be very small for nonvolatile compounds such as lead oleate, lead acetate and lead arsenate. Volatile compounds, notably lead tetraethyl, penetrate the skin with great ease.[28] The maximum allowable concentration for lead, which has been standardized for some years at 1.5 mg. per 10 cubic meters of air, has been criticized as not always applicable.[29] Insoluble lead compounds such as lead chromate are definitely less toxic, and review and standardization of each type of lead compound has been suggested. Experimental studies on the use of BAL, which had been found efficacious[30] in arsenic and mercury poisoning, showed that although it increases lead excretion, it apparently has no effect on decreasing the toxicity of the absorbable lead. Other lead antidotes, specifically a soluble dithizone, are being studied. A spectrophotometric method for rapid lead urinalysis shows great promise as a screening technic for lead absorption.[31]

Considerable attention was paid to the carcinogenic properties of various industrial chemicals. Evidence is accumulating rapidly that exposure to chromates greatly increases the incidence of pulmonary and other carcinomas.[32] The number of cases of cancer in a factory handling arsenic indicates that there is an excess of deaths from this cause in such workers.[33] Carcinomas of the bladder from aniline do not occur because exposure is sufficiently well controlled, but beta-naphthylamine and benzidine still produce cases.[34, 35]

Princi[36] doubts that cadmium, which is considered a severe industrial toxin, is capable of producing chronic effects. Friberg,[37] however, describes kidney damage in a group of workers exposed to nickel and cadmium, although previous reports in the literature give no direct evidence that either of these metals causes pathological changes in the kidney. An important contribution to the toxicity of sodium fluoride has been made by the Public Health Service[38] in a study of exposure at open-hearth furnaces. Irritating upper respiratory symptoms may occur, but there is no evidence of any pulmonary cardiovascular or skeletal findings of fluorosis. Also, no effect was noted on the teeth or tissues of the oral cavity in workers using sodium fluoride.

Hardy and Elkins[39] reached the conclusion that the maximum allowable concentration for benzene

should be reduced from 75 to 35 parts per million, and Forssmann and Frykholm[40] state that exposure to benzene creates an increased demand for vitamin C and that an extra supply increases the resistance to the effects of this toxin. Clinton[41] describes an accident in which a large amount of selenium was released in the air. No systemic toxic effects were noted, and only severe local irritation occurred. A comprehensive review of the acute effects and treatment of chlorine inhalation was described by Chasis et al.[42] when over 400 people were accidentally poisoned with this gas after rupture of a chlorine tank. No chronic pulmonary sequelae were noted in any of the patients who were followed for sixteen months although some had persistent anxiety reactions.

Dermatitis

With occupational dermatitis constituting approximately 65 per cent of all cases of occupational diseases,[43, 44] the dermatologic problem continued to demand constant vigilance and study on the part of the industrial physician. No outstanding developments were reported in this field, but over-all methods of control were reviewed by a number of authorities. All were agreed on the importance of preventive measures such as provision of proper cleansing facilities, substitution of nonirritating materials when possible, use of protective clothing and creams, and careful placement of employees.

Criteria for determining the industrial status of skin diseases were discussed by Bulmer,[43] Macauley,[44] Downing[45] and others. About 40 per cent of a large number of cases of employees claiming compensation for occupational dermatitis, on which Downing reported, were proved to be of nonindustrial origin. Approximately 20 per cent or less of industrial skin diseases are allergic, according to Tolmach,[46] but the percentage was difficult to determine since diagnoses were frequently based on inaccurate histories or solely on patch tests, improperly applied.

The skin hazards in 114 occupations and the newer therapeutic agents, as well as dermatoses due to wearing apparel and to various cosmetic preparations, were considered by Schwartz et al.[47] In the last-named category, a cause of dermatitis, mentioned elsewhere,[48] is prolonged or frequent contact with the so-called "cold" permanent hair-waving preparations. Employees' exposures to skin hazards away from work, as well as on the job, should be taken into account before the physician renders his final opinion about the etiologic agent in each case.[49] Investigations were reported on different occupations, such as flour allergy with cutaneous reactions,[50] paint-factory workers and painters with occupational dermatoses,[51] workers exposed to pitch, tar or tar products[52, 53] and farmers and others suffering from grain itch.[54, 55] Other occupations studied for their skin hazards were those of longshoremen[56] and workers in the jewelry industry[57] and in the poultry industry.[58]

Numerous procedures for the control of specified occupational skin hazards were recommended. For employees working with chromium salts, the use of a nasal paste of 5 per cent bismuth subnitrate in soft paraffin was found effective to prevent perforation. This method was adopted in German chromium plants, where 30 to 40 cases of lung cancer had occurred.[59] Reviewing the poisonous properties of DDT, Van Huyt[60] advised the wearing of synthetic-rubber gloves and provision of good ventilation where it is being used. Solvents as vehicles for DDT were studied and reported on from the standpoint of their photosensitizing action on the skin.[61] Carbitol, which is being used more and more in cosmetic or other preparations for the skin, can constitute a health hazard, if uncontrolled.[62] Temporary loss of hair among workers in neoprene-manufacturing plants occurs only in the polymerization areas, it was found.[63] No satisfactory treatment or protective headgear was developed, but the condition was controlled or eliminated by frequent and complete changes of air in the polymerization areas. In general, precautions should be taken not only in the particular location where any irritant fume or solvent mist is present but also in nearby areas to which air currents can transport such agents, according to Campbell,[64] who observed several cases of dermatitis due to this indirect type of exposure.

The testing of a large number of protective-glove materials for their permeability to tetraethyl lead and ethylene bromide led Calingaent and Shapiro[65] to conclude that nylon and also nylon compounded with neoprene are the most impermeable and best suited for this purpose. Various barrier creams were investigated by Cumming[66] and others, with a view to lowering the high incidence of dermatitis in industries manufacturing explosives, particularly tetryl and other nitrobodies. A new protective cream, with liquid paraffin forming the basis, was developed. Other barrier creams, for workers handling the soluble phenols and cresol formaldehyde products, and for workers exposed to pitch, were reported by Weldon.[59]

Rehabilitation and Job Placement

The proposition that rehabilitation of the disabled worker is a vital function of industrial medicine is being accepted more and more widely, here and abroad. The need for veterans' rehabilitation programs had been recognized and acted upon early in the post-war period, but until more recently such programs were only very rarely extended to include the physically impaired among civilian industrial workers. That a large potential of human productivity was being by-passed by industry — to the detriment of industry itself, as well as of the community at large — was frequently noted. To overcome the resistance of industry in accepting phys-

ically impaired workers because of increased liabilities under the workmen's compensation laws, New York State amended its Second Injury Fund to provide equitable adjustment of the liability an employer assumes in the employment of a worker with any permanent physical impairment.[67]

Hanman[68] observed that the physically handicapped comprise a majority of the population. He pointed further to the numerous studies that have shown that disabled employees, properly placed in industry, can be equal or superior to others. This estimation was proved correct in various reports on actual experience in the selective placement of the so-called unfit. The New Jersey Rehabilitation Commission[69] and the Federal Bureau of Labor Statistics reported that experience has shown the physically handicapped to be more efficient, to lose less time away from the job, to have more social contacts and to be less prone to accidents (except for the hard of hearing) than other employees.

Opinion about the ideal type of organization and sponsorship of rehabilitation programs varied. Rehabilitation centers supplementing hospitals and set up on a community basis were foreseen for the future by Deaver,[70] and the present lack of such centers was deplored by Rusk[71] and various others. A co-ordinated rehabilitation bureau to serve the entire district of Lyons, France, was proposed as a model by Gauthier et al.[72] That society has an undeniable responsibility toward the economic and social restoration of the handicapped was not disputed, but industry's share in this responsibility was questioned. Management's hesitancy to hire workers not in perfect health was considered valid by Luongo,[73] who pointed to the risks of subsequent unmerited compensation appeals, especially in cases of tuberculosis, silicosis and mental disorders. On the other hand, Pringle[74] expressed a broader concept of industrial health as indivisible into "health-at-home" and "health-at-work" categories.

The practical benefits in industry's employment of the handicapped were publicized by a number of organizations, such as the National Association for the Employment of the Handicapped and the Office of Vocational Rehabilitation of the Federal Security Agency.[75] It was estimated that approximately 8,000,000 men, including veterans and civilians, would require the services of rehabilitation and vocational-guidance centers.[71] Yet, rather than increasing, the number of those applying to existing rehabilitation centers for training and jobs decreased.

A prerequisite in any industrial rehabilitation program is the co-operation of labor, industry, medicine and sociology,[76] but rehabilitation is primarily a function of surgical and medical care.[77] Close co-ordination between job assessor and physician is necessary, according to Lloyd,[78] who gave a detailed description of the job analysis method used in matching the rehabilitated worker to the job, as well as a rapid and practical system of recording the analytical data. For workers injured in industry, Aitken[79] found that aftercare, with a view to the patient's re-employment, has been generally inadequate.

Attention was focused on the rehabilitation needs and potential productivity of persons disabled by specified illnesses or injuries: tuberculosis, cardiac conditions, rheumatic ailments and loss of limbs. Sander,[80] in a report on tuberculosis in industry, showed that it was unnecessary to refuse employment to job applicants with a history of this disease. He presented a guide for establishing the worker's clinical status and effecting proper placement and follow-up examinations. The difficulties encountered by persons with arrested tuberculosis were described by Moller.[81] In workers with heart disease, the cardiac clinic and employment service should co-operate to appraise each patient's physical capacities, analyze the physical demands of the job and match the latter against the former.[82] Sprague[83] advised complete cardiac studies of cardiac patients before employment is considered and regular reclassification checkups with the use of electrocardiography for those already employed. The training and placing in suitable work of rheumatic patients was discussed by Bonni,[84] and in another report the problem of dealing with injuries in workers affected by arthritic conditions was explored.

In the rehabilitation of civilians with amputations Kessler[85] stressed the lack of, and great need for, a program like that developed by the armed forces. The lack of such a service for persons disabled in industrial accidents was noted by Devesa[86] in Spain, where the patient is offered the alternative of a cash settlement or a prosthesis of poor quality and usually chooses the former.

Psychiatry

Investigation in the field of industrial psychiatry suggested vast possibilities for the future. The urgent need for wider application of the psychiatric methods already proved and for further study and greatly expanded training programs was repeatedly stressed. Several surveys showed a direct relation between the mental and emotional health of workers and their proneness to frequent accidents, general unrest on the job — as evidenced in lateness and absenteeism — and even physical illness. Hence, attention to the psychologic impairment of employees was advocated as a cost-saving necessity for industry, if not as an obligation to the community.

Without proposing specific methods of administering psychiatric services, numerous spokesmen for industrial management and the medical profession urged consideration of human relations and of the emotional element involved in industrial employment.[87, 88] Striking proof of the importance of psychologic factors in industrial efficiency was shown in a detailed study of the incidence of neuro-

sis in factory workers, the factors predisposing to it and its effects on production, reported in England.[89] Of 3000 workers, 10 per cent had suffered definite neurosis and 20 per cent had suffered minor neurosis. Neurotic illness caused from a quarter to a third of all absence due to illness. The same frequency was found in workers at higher and lower levels of skill and earnings. The incidence was greater among women than men. The survey showed that neurosis was most common among workers with few social contacts and unsatisfactory domestic circumstances, and especially among those who disliked or were bored by their jobs. Fatigue, inadequate diet and lack of proper lighting on the job contributed to neurotic illness.

That mental incapacity in industry is related to group morale was established by Brodman and Hellman,[89] who found that frequent short illnesses occurred in departments where frequent lateness — a concomitant of poor morale — was also noted. The data further brought out that the individual's work morale is definitely influenced by the group and that different work groups have dissimilar morale.

Eliminating basic causes of employee maladjustment would do away with many grievances that start as minor complaints.[90] The role of the individual employee's supervisor or foreman in this type of adjustment was discussed by Brodman,[91] Marrow,[92] Maier[93] and others. Training programs were suggested to bring to first-line supervisors an awareness of, and ability to meet, the underlying complicated problems of emotionally disturbed workers.

Mounting concern for the costly effects of alcoholism among industrial employees was revealed in the growing number of studies on the subject. Bacon[94] estimated that of approximately 4,000,000 alcoholic persons in the United States, about 2,890,000 are men between the ages of thirty and sixty, and about 1,370,000 "chronic, excessive and compulsive drinkers are fairly regularly employed in industry." The problem is more susceptible to correction in industry than in the community at large and can be met with the help of psychiatry.

The industrial physician must bring himself up to date psychiatrically, according to Moorad,[95] who quoted statistics to show that from 85 to 90 per cent of accidents are due to "human factors." Healthy workers with personality disorders should be the responsibility of industry, for its own benefit. They not only have repeated accidents, Moorad declared, but also contribute heavily to absenteeism and project their dissatisfaction and low morale among their fellow employees. He described five prominent types, showing how and why each can cause accidents. The first step, in any case, is proper placement or elimination, but workers with neurotic tendencies should not be eliminated entirely, since cure and adjustment are possible through psychiatric treatment.[96] Patients with neurosis and employment difficulties have been rehabilitated with notable success at a unique industrial neurosis unit at Sutton Hospital in England.[97]

Reports on various procedures used in placing and counseling employees indicated an increasing interest in this aspect of industrial psychiatry. The Rorschach (ink-blot) test is a valuable supplement to the customary testing and interviewing technics. Noting that 1 out of every 11 employees have serious temperamental difficulties, Armbruster[98] held that job applicants should be tested not only for aptitude but also for interest in the work, motivation, general intelligence and temperament. The use of employee tests should be accompanied by an understanding of "the dynamic aspects of personality problems and of the complicated processes involved in group adjustment."[99]

Counseling technics were discussed by Himler,[100] who observed that "skilled listing . . . constitutes the major part of the counselor's therapeutic armamentarium." In another report, industrial nondirective counseling was found to have advantages over clinical nondirective counseling, because of the more casual and intimate contact in the plant as compared to that found in clinical psychotherapy.[101]

Chronic industrial fatigue was seen as a contributing factor to emotional instability, absenteeism and proneness to accidents. The installation of "color conditioning," making scientific use of wall and fixture paints, was reported in several cases to result in less eyestrain and fatigue and greater efficiency,[102] whereas noise control was given as another means of reducing mental fatigue and abnormal emotional reactions.[103] In discussing industrial fatigue, Carozzi[104] argued that more important than adapting machine and working conditions to the worker was attention to him as a human being, in his personal, family and broader social settings.

A notable step forward was seen in the inauguration of a pioneer program to train psychiatrists for the industrial-relations and labor field at Cornell University.[105] At the same time, at Harvard University and Massachusetts Institute of Technology scientific investigation was under way into the social— as distinguished from the physical — working environment of industrial employees.

Education

Educational facilities for physicians intending to enter the industrial service were extended here and there, by a few universities, and with the co-operation of certain local or state medical societies, hospitals and industries, but a general, standardized program was still lacking. However, progress was made in orienting individuals and organizations, both professional and industrial, toward accepting the inevitability of specialized industrial medical education. With emphasis shifting toward the preventive and research aspects of the field, there was

little question that more trained and experienced occupational practitioners were needed.

Medical educators, in large measure, have failed to adapt their training or practice to meet the needs of the present-day American community, Kehoe[106] pointed out. No comparable field of medicine has offered so few opportunities for formal training as that of industrial hygiene.[107] Lacking specialized educational facilities, industrial medical directors "have evolved via the school of experience from general practitioners, general surgeons and internists," Brown[108] declared. Because well trained industrial medical men "are not to be found," he added, "there are . . . an enormous number of small plants of every description without any medical department much less a health program."

There must be accord among industrial physicians, themselves, on the scope, objectives and methods of their practice, before teaching centers can be expected to provide the necessary instructional facilities, Buchan[109] asserted. Industry too "must make known in no uncertain terms its needs in medical and ancillary personnel, its needs in applied and fundamental industrial medical research."

According to several proposed plans, graduate work should include, as an essential, in-plant service or residency. One large industry's in-plant fellowship program, conducted with the co-operation of a leading medical school and said to be the first of its kind in this country, was described.[110] In most of the graduate courses now offered, "a major defect . . . is the lack of opportunity to give an adequate on the job experience."[107]

In the undergraduate curriculum, Kronenberg[111] finds that "industrial medicine must be introduced and expanded . . . if medical education is to be progressive and useful and meet present-day needs." Certain groundwork material can be introduced by "infiltrative tactics" in preclinical departments, Goldwater[112] stated, but industrial medicine, if regarded as a specialty, "obviously calls for special graduate or postgraduate training." He suggested that schools of public health were appropriate centers for training industrial physicians, as well as industrial nurses, engineers and chemists, and cited the inauguration of such a program at Columbia University. Among the nation's nine schools of public health, courses in industrial health and opportunities for advanced specialization in the field are generally provided.[113]

A crucial factor in the solution of the educational problem is the lack of established standards. Some progress was shown in the fact that residencies in the specialty of occupational medicine have been placed on a formal basis comparable to other residencies in line with the plan adopted by all certification boards.[114] The chief difficulty remaining is to obtain suitable applicants and establish the fellowships themselves. From the viewpoint of the prospective candidates in the field of industrial medicine both inducement and financial means were seen to be lacking.[115] The failure of the medical profession to dignify industrial medicine with official recognition as a specialty was considered a deterrent to students in deciding to enter the field. Scholarship and fellowship funds are being established, and industry itself has been urged to subsidize graduate training.[116]

Medical Services

Marked inequalities persisted in the medical services provided for industrial workers in different localities and from one industry to another. In general, in-plant medical services were maintained and continually improved upon by large industries, but were still inaccessible to small industries. To what extent this disparity affects the whole picture can be judged by the fact that small concerns are estimated to constitute 99 per cent of the nation's industrial plants,[117] employing about 60 per cent of the total labor force.[118]

No country-wide studies of the situation were reported on, but several surveys of limited groups of representative plants were made. One such study of 565 plants employing 500 or fewer workers showed that only a few provided even a minimum of medical care.[117]

In Illinois it was found that more than 12,000 small industrial establishments had no medical service,[119] whereas in Connecticut,[120] 1400 plants employed a total of 119 physicians.

Diverse approaches to the small plants' health problem were recommended. In France[121] small businesses affiliate to form a joint medical unit, a system successfully tried in one Pennsylvania community.[122] Management-labor "safety committees"[123] have been organized in industries of all sizes in Sweden and also have functioned satisfactorily in some small or medium plants here.[124] In one plant of less than 1000 employees a single, full-time male nurse effectively directed the combined health, safety and welfare departments.[125] Regional programs were instituted, mostly advisory in character, in various centers such as Chicago[126] and Washington, D. C.,[127] and in areas of Wisconsin,[128] Indiana[129] and Nebraska.[130]

A total of 58 industrial-hygiene units were functioning under the Industrial Hygiene Division of the United States Public Health Service, with 43 per cent of the budget deriving from state and local funds and the remainder from federal funds.[131]

Among new types of service reported from abroad was that in Manitoba, Canada.[132] An industrial-hygiene bureau, operating in a comparatively thinly populated region, attempted to improve the health conditions in small plants and conducted a program featuring portable x-ray equipment, plant surveys and stipple-cell counting service. For railway workers in France, four trains fitted with x-ray apparatus were traveling all over the country and

stopping wherever the equipment was needed for employees' periodic physical examinations.[133]

The extension of medical-service programs to all industries was urged not only on humanitarian grounds but also on the basis of increased production. On-the-job medical care was estimated to reduce absenteeism due to sickness up to 60 per cent.[117]

The expansion of existing services in larger plants included the building of new dispensaries and the addition of various specialists to the medical-department staffs. Periodic physical examinations and special checkups for workers in hazardous areas were increasingly the custom.[134]

Heightened interest in industrial eye and dental programs was expressed.[135, 136] Teamwork of all those responsible for the employees' welfare was declared essential in the provision of health services. Derryberry[137] called on industrial physicians to realize "that the medical sciences are but a part of the total technology which must be brought to bear on the maintenance of health and the prevention of disease and disability."

The value of proper record keeping in industrial medical departments was frequently stressed and various suggestions for improved recording systems were offered.[138-141]

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

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CASE 35131

PRESENTATION OF CASE

A thirty-six-year-old woman, a clerk, entered the hospital with a chief complaint of increasing constipation.

Six years prior to admission, because of right-lower-quadrant "gripping" pain and abdominal signs (details unknown), she had an exploratory laparotomy, with excision of a right ovarian cyst and a normal appendix. For two years following this procedure she was asymptomatic, but then the right-lower-quadrant pain began to recur several times a week. Two years before admission she began to notice midsacral pressure discomfort at the time of her menstrual periods, with partial relief by bowel movements. During the year before entry she had occasional sharp left-lower-quadrant pain with her menses. One and a half years before admission, for a period of five days, she had very black vaginal bleeding followed by her usual menstrual flow, and four months later three episodes of vaginal bleeding in one month. Otherwise, the menstrual periods were regular, occurring every twenty-four to twenty-six days and lasting five days, with a normal quantity of flow containing some clots. For two years prior to admission she was increasingly constipated, except that at the time of the menstrual periods, the stools were very loose and occasionally quite dark, but never black or bloody. She had some mild gaseous indigestion, without specific food intolerance or relation to meals. There was no anorexia or weight loss, and the system history was not remarkable. During the year and a half before admission three barium enemas were done elsewhere. The last two of these examinations allegedly showed a polyp in the sigmoid.

Her mother had diabetes, and a mastectomy (diagnosis unknown) had been performed.

Physical examination disclosed a normally developed and well nourished woman in no discomfort. There was a well healed right-lower-quadrant paramedian scar. To deep pressure there was tenderness in the right upper and both lower quadrants, with the maximal tenderness in the right lower quadrant. The sigmoid was palpable in the left lower quadrant and was moderately tender. There was no distention, and peristalsis was normal. Pelvic and rectal examinations were entirely negative.

The temperature, pulse and respirations were not remarkable. The blood pressure was 110 systolic, 70 diastolic.

The urine examination was negative. The hemoglobin (photoelectric-cell technic) was 14.5 gm., and the white-cell count was 7500. A blood Hinton test was negative. A barium enema demonstrated a rounded, filling defect, 2 cm. in diameter, in the sigmoid at the junction with the descending colon.

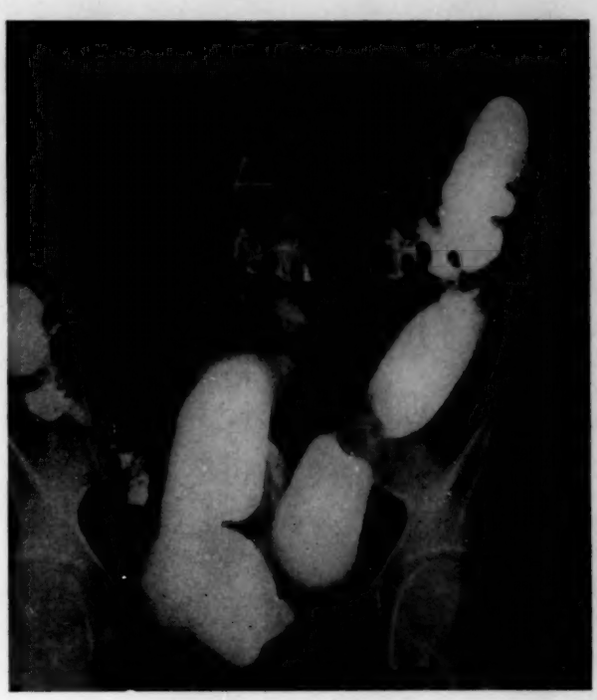

FIGURE 1.

The bowel in the immediate vicinity of the defect was smooth in contour, and the lumen was narrowed (Fig. 1 and 2). There was some suggestion that with pressure, a change in the defect could be produced. Following evacuation, air was injected into the large bowel in an attempt to disclose any other polypoid defects. No other definite masses were made out, although at about 8 cm. from the lesion described there was a questionable defect, about 5 mm. in diameter, in the bowel that was partially outlined with barium. The remainder of the colon was not unusual.

After preparation with sulfasuxidine by mouth, an operation was performed on the fifth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. CLAUDE E. WELCH: May we see the x-ray films?

DR. STANLEY M. WYMAN: On all the films there is a constant, round, smooth defect overlying the brim of the pelvis in the wall of the ascending colon, near the junction of the sigmoid. This filling defect appears perfectly smooth, and there appears to be a preservation of the mucosa over the lesion. On some of the spot films it has a suggestion of a hook, proximally and distally, that we see with intramural, extramucosal lesions. This film has a suggestion of a small, less definite mass just above the primary mass on the opposite wall of the bowel, but it is not well seen on all films, although it is seen on this large film of the colon. I should prefer to call this

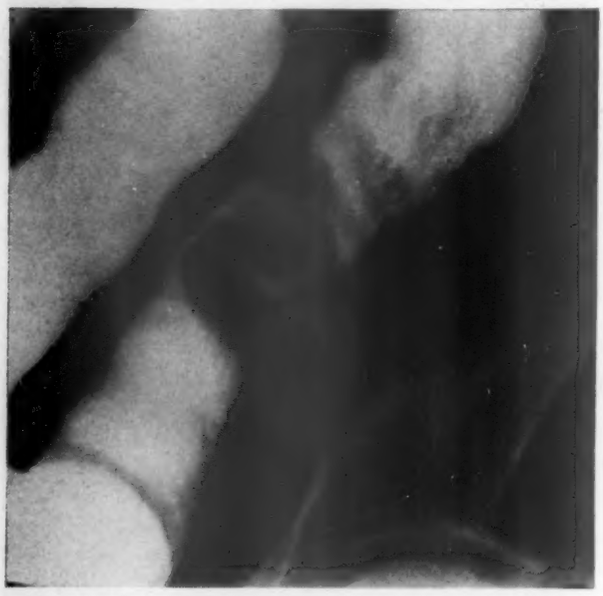

FIGURE 2.

an intramural extramucosal mass rather than a polypoid tumor of the colon, although one cannot be entirely certain.

DR. WELCH: I think the discussion of this case will be more interesting if I make a diagnosis first and then attempt to support it by the data we have. This ought to be an endometrioma of the sigmoid. If it is not, it still should be.

Let us go back over the history to see whether we can substantiate that diagnosis. At operation six years before entry a right ovarian cyst and a normal appendix were excised. Two years thereafter the patient was asymptomatic, but then the right-lower-quadrant pain began to recur. The next question arises, Could the pain have been associated with the lesion now discovered in the sigmoid? I am inclined to think that it was not pain in the right lower quadrant associated with sigmoid disease. This type of pain has been described frequently, but the right-lower-quadrant pain from primary sigmoid disease is usually due to distention of the cecum from a constricting lesion in the sigmoid or from a contiguous, inflammatory spread from an inflamed sigmoid that has prolapsed over into the right lower quadrant. There is no evidence of distention or inflammation of the bowel, and so I assume that there was something else in the right lower quadrant. She may have had excision of an endometrial cyst at the first operation, with retention of a portion of the ovary, and again some endometriosis had developed in the remaining portion. It is surprising to find that she did not have more menstrual pain; this would indicate, if this diagnosis is correct, that endometriosis in the pelvis was not terribly extensive. The black bleeding suggests that she had adenomyosis of the uterus as well.

I assume that a proctoscopy was done and was negative.

DR. BENJAMIN CASTLEMAN: That is right.

DR. WELCH: I assume that there was no actual bleeding from the rectum at the time of the period. That might be of interest to know. Endometriomas rarely involve the mucosa, but occasionally do, with bleeding by rectum. The defect in the sigmoid on x-ray examination is perfectly compatible with this diagnosis. We have had in this hospital several lesions in the lower descending colon or upper sigmoid at this level that have been endometriosis. Interestingly enough, some have not become symptomatic until after the menopause. I suppose the increasing fibrosis that takes place after atrophy of the endometrial tissue produces obstruction. This lesion can be assumed to be still active, and the increase in symptoms at the time of the periods is explained by the congestion that occurs in the tumor. Endometriomas in the gastrointestinal tract are found chiefly in the sigmoid or on the surface of the appendix and very rarely in the lower ileum or cecum. The position of this lesion is consistent. The negative pelvic examination indicates that disease in the other pelvic viscera is minimal.

I imagine the surgeon on entering the abdomen was confronted with a minor amount of endometriosis in the ovaries, a small segment of remaining right ovary and a lesion in the sigmoid, about which it was impossible to tell the exact nature. The statement has been made in the literature that when endometriosis is the diagnosis the patient should preferably be treated by sterilization, after which the lesion will atrophy and the dangers of bowel resection will be avoided. That, I assume, was not carried out in this case for two reasons. In the first place, no one knows what the lesion is, and even if atrophy of the endometriosis did occur, there is so much involvement of the sigmoid that it is likely to progress to obstruction later on. Consequently, I assume that a resection of

the colon was done in this patient. Whether or not a hysterectomy should have been done as well would have to be determined by the findings at the time of operation.

Dr. Leland S. McKittrick: Dr. Welch has been very logical as well as practical in his reasoning. Dr. West, the resident, also made a preoperative diagnosis of endometriosis on this patient. When the abdomen was opened I did not know what it was. I thought it was carcinoma. The lesion was just as hard as it could be. It had a characteristic appearance in the gross of a small, constrictive, scirrhous type of carcinoma, with an epiploic appendix adherent over the top of it. During the course of the resection Dr. Risley, who was helping me, called attention to a small nodule along the bowel, about 6 or 7 cm. from the primary lesion. This was a whitish, hard area, probably 3 mm. in diameter, which looked for all the world like a carcinomatous implant. It did not have the color that one associates with an endometrial implant, and that led us to think that this was probably carcinoma rather than endometrioma. So we broadened the amount of bowel we took out. We took out more bowel than I would have taken out if I had been confident of the diagnosis when we operated because I honestly thought that we were dealing with carcinoma of the sigmoid. The patient did have a cystic tumor or mass, about 6 cm. in diameter, on the left ovary that no one had felt prior to operation. She did not relax too well; I think that would have been felt if she had been examined under anesthesia or had relaxed well.

Clinical Diagnosis

Carcinoma of sigmoid.

Dr. Welch's Diagnosis

Endometrioma of sigmoid.

Anatomical Diagnosis

Endometriosis of sigmoid.

Pathological Discussion

Dr. Castleman: The lesion that Dr. McKittrick described in the upper sigmoid presented on the serosa as a puckered, white, constricting process, which on section was seen to extend deep into the muscularis. It was grayish white and fibrous rather than granular as is seen in carcinoma. The mucosa over it was normal. The lesion measured about 3 by 2 by 1 cm., and microscopically was characteristic of endometriosis.

The microscopical section (Fig. 3) showed how deep the endometrial glands had extended into the muscle layers. In a few foci the process was very close to the mucosa. A few centimeters above this lesion was another smaller one, which corresponded to the upper defect seen on the roentgenogram. There are, as Dr. Welch stated, some surgeons who believe that even if the patient is known to have endometriosis, the bowel should be resected in the presence of obstruction. Three years ago at these exercises in the discussion of a case of endometriosis, I made the statement that when endometriosis involves the sigmoid, resection has often been necessary.* Dr. James R. Miller, of Hartford, wrote in and questioned the need for resection of the bowel for endometriosis, believing that castration would produce regression of the sigmoid lesion. I agree that this probably would occur in some cases,

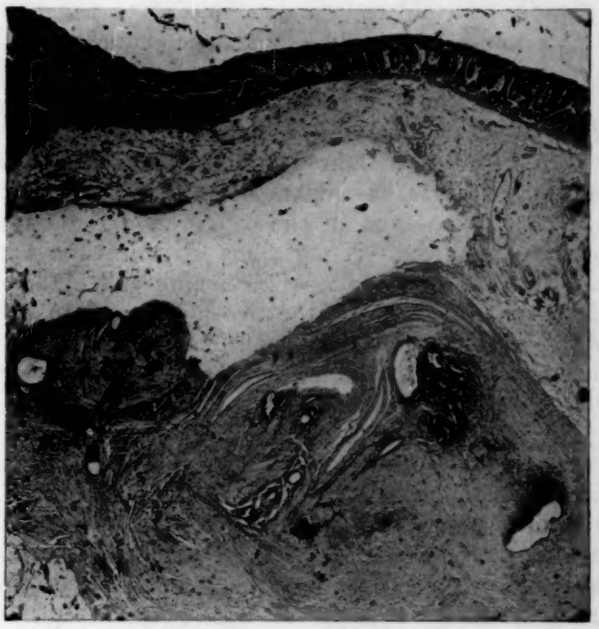

Figure 3. *Photomicrograph of Sigmoid, Showing Endometriosis in the Muscularis Close to Edematous and Fragmented (Artifact) Submucosa.*

but in a case like this, with the disease extending so deep, it may be too late. Then there is the point that Dr. Welch makes: that after the menopause the lesion may constrict even more.

Have you had any experience with that, Dr. McKittrick?

Dr. McKittrick: The first one I did was on a woman sixty-five years old. I do not believe the ovaries were doing much functioning, and she was operated on because she was obstructed. But going back to the case we are discussing, I believe that if I were in this woman's place I would rather have ovarian tissue and have had a segment of bowel resected than to have the bowel not resected and be sterilized at thirty-six years of age.

Dr. Castleman: As long as you could do an end-to-end anastomosis.

Dr. McKittrick: That is correct. This patient had not had much discomfort. She was only thirty-

*Case Records of the Massachusetts General Hospital (Case 32321). *New Eng. J. Med.* **235**:199-201, 1946.

six, and her right ovary and tube had been taken out. She was young, I think, for one to go ahead and do a complete castration job unless it had been necessary to do so. It seemed to me that if she progressed along reasonably well, the longer a definite operation was postponed the more freedom we would have to do the thing when it had to be done. She has convalesced and gone home.

CASE 35132

Presentation of Case

A fifty-year-old man was admitted to the hospital because of jaundice and a weight loss of 26 pounds.

Three months before admission he had an attack of severe, sharp, right-upper-quadrant pain, radiating to below the right scapula. This pain was crampy and was accompanied by distention. He was hospitalized and in the course of a few days became jaundiced. X-ray films at this time are said to have shown a nonfunctioning gall bladder and a gastric ulcer. In the two months preceding admission to this hospital the intensity of the jaundice gradually decreased. He lost 26 pounds in weight during this period.

The patient had had indigestion relieved by either food or water since boyhood. Twelve years before admission a perforated duodenal ulcer was sutured, and since then he had followed a strict dietary regime. He continued to have "ulcer pains," but there had been no hematemesis or vomiting. Eight years before admission he was hospitalized for "stoppage of the bowels" relieved by laxatives and enemas.

Physical examination showed faint jaundice still present in the scleras. The heart and lungs were normal. There was a smooth, firm, tender, movable mass, about 3-4 cm. in diameter, in the right upper quadrant, which descended with respiration. This was thought by all the examiners to be the gall bladder. The liver was not palpable.

The temperature, pulse and respirations were normal. The blood pressure was 130 systolic, 90 diastolic.

Examination of the urine revealed a specific gravity of 1.022, a + test for albumin, negative tests for sugar and diacetic acid and a +++ test for bile. Examination of the blood disclosed a hemoglobin of 12.2 gm. and a white-cell count of 13,300, with a normal differential. The nonprotein nitrogen was 23 mg. per 100 cc., and the total protein 7.35 gm. (albumin-globulin ratio of 1.8); the serum bilirubin was 4.1 mg. per 100 cc. direct and 4.6 mg. indirect, and the alkaline phosphatase 28.9 mg. A cephalin flocculation reaction was + in twenty-four and ++ in forty-eight hours, and the thymol turbidity 3.0 units. A blood Hinton test was negative. A gastric analysis showed free hydrochloric acid and a negative guaiac reaction.

A Graham test demonstrated failure of the gall bladder to fill. In the right upper abdomen there was a dense calcification, 1.0 cm. in diameter, that was consistent with a stone in the gall bladder or common duct or mesenteric lymph node. A cluster of calcifications, probably in the pancreas, was also noted. A barium enema was negative. A gastrointestinal series was done. The stomach was pliable, and its mucosal pattern within normal limits. The patient permitted little palpation in this region, but no ulceration or filling defects were demonstrated. The duodenal bulb was deformed in a manner consistent with previous surgery. There was an irregularity of the lesser curvature and of the duodenal loop about which no opinion was given.

On the eighth hospital day an operation was performed.

Differential Diagnosis

Dr. Chester M. Jones: This is the sort of case in which a precise diagnosis is interesting but unimportant. The important thing is the decision what to do, and the sooner that decision is made, the better. One may make an intelligent guess that is logical but a mistaken diagnosis. The past history was that of ulcer with perforation twelve years before admission. The perforation was probably closed successfully. The duodenal-ulcer problem remained. Closure does not solve that problem at all. The episode eight years prior to admission, which was called intestinal obstruction and which responded easily to simple methods and did not recur, is not pertinent to the present discussion. The subsequent history of prolonged change in the bowel habit, I judge, was due to fecal impaction.

That leaves us with a man of fifty years with a three-month history initiated by right-upper-quadrant pain, which is in the classic location for gallstone colic. The pain radiated through to the right scapula, following which he was jaundiced. On the basis of the story alone and in the absence of other demonstrable facts, there is no reasonable doubt that he had biliary colic. The fact that a gastric ulcer was said to have been demonstrated at this time is interesting, but it is hearsay evidence. It may have been a faulty interpretation. It would not be the first time that a gastric deformity, even one that persisted for an unusual period, was not found at exploration, but at operation gallstones were present. In other words, for some reason or other, gall-bladder disease associated with stones can occasionally produce a constant gastric deformity that is indistinguishable from cancer or ulcer. It is very uncommon but it does occur, and I can think of several important cases here in which the diagnosis prior to operation was gastric cancer and at operation we found only gallstones.

The diagnosis of gastric ulcer was not sustained when a probably adequate examination of the stomach was made. A duodenal lesion was demonstrated after admission to the hospital. Therefore, we can put a question mark against the original suggestion of gastric ulcer. If he did not have gastric ulcer, which always means that the ulcerating lesion may be cancer, we can focus our attention on the biliary tract as the basis of the symptoms three months before admission. The x-ray film, which I have not seen but would like to see shortly, was certainly entirely consistent with biliary disease. He could have had a stone either in the gall bladder — with a resulting gall-bladder infection and an acute cholecystitis, in addition to a cholangitis — or in the common duct with a ball-valve action and jaundice with an attack. That diagnosis is tenable only to a certain point and may fail to explain what subsequently happened. He had lost 26 pounds in weight in the presence of diminishing jaundice. As far as I can tell from the history, there was no particular pain or discomfort associated with it. He did not vomit. There is no explanation given for this loss of weight, but it is a striking fact observed over a relatively short period. If he had had fever, one could explain the loss of weight on that basis. Certainly, an attack of biliary colic due to stone three months previously should not produce a 26-pound weight loss, without continuous or added symptoms.

Physical examination again directs attention to the biliary tract, and the statement is made that several examiners felt a tender, firm, smooth mass. It is not said whether the mass was lobular or round, which one would like to know. The mass was felt in connection with the liver below it. It moved with respiration, and everyone thought it was a palpable gall bladder. A palpable gall bladder in the absence of jaundice immediately following an acute attack of biliary colic can be due to hydrops with stone in the ampulla of the cystic duct. It very frequently occurs as a transitory phenomenon, present for a few hours and then disappearing as the stone moves. A gall bladder palpable three months after the only attack of biliary colic probably should be explained on some other basis. This is not a good example of Courvoisier's law in the sense that it cannot be described with painless jaundice. The jaundice nearly disappeared, and there was no pain for three months. He had not had anything else for three months. The jaundice was out of the picture except at a minimal degree. At the present time one can at least invoke Courvoisier's law and try to play with it a bit in this sense: here was a patient with a palpable mass thought to be gall bladder, and he had jaundice. That should mean ordinarily a pathologic gall bladder and obstruction at the ampulla of the common duct. That is the real meaning of Courvoisier's law. Usually the obstructing lesion is cancer of the head of the pancreas or of the ampulla or of the bile duct. The burden of proof is usually on the man who says it is not carcinoma. Rarely, we do have a palpable gall bladder in the presence of jaundice due to common-duct stone. It is very uncommon, although we have a few cases as every hospital has.

In this case there may be plugging of the duct due to a growth. There may be external pressure, let us say from a malignant lesion, which is causing marked lymphadenopathy with obstruction of the common duct at a certain level. The evidence that he had infection in the liver itself, I would say, is negligible. He could have had pain and jaundice with acute epidemic hepatitis. That is fairly common. The jaundice could persist for three months, but he should not have had a palpable gall bladder, and the liver-function tests should not have been as described in this case. He should have had a high cephalin flocculation, high thymol turbidity and a low white-cell count rather than a high one, and the differential should not have been normal. It does not seem logical to interject that possibility into the picture at all. Therefore, it seems to me that we have a right to consider one of two things without observation of the x-ray films at the moment: either a gallstone with or without a stone in the common duct, which I think is relatively unlikely, or a malignant lesion in the region of the ampulla of Vater, in the pancreas, in the ampulla itself, in the bile duct with gradual spread to the adjacent areas or even in the gall bladder.

My own belief at this stage is that the patient had neoplasm in the neighborhood of the ampulla as well as gallstones, and I am sure he ought to have been explored on the basis of this protocol alone.

I should like to see the x-ray films. They can modify the diagnostic possibilities but not the approach that should be taken.

Dr. Joseph Hanelin: The initial examination, a cholecystogram, shows a nonfunctioning gall

bladder and a calcium density, 1 cm. in diameter, in the upper abdomen. In addition, there are multiple punctate calcifications, which extend from the right aspect of the second lumbar vertebra upward and to the left. These are very likely pancreatic calcifications. The larger calcified density in the upper abdomen is seen to overlie the spine and may represent a stone that had its origin in the gall bladder. It is situated somewhat higher and more medial than the usual position of the gall bladder so that it might be within the cystic or common duct.

DR. JONES: It is fairly high to be in the ampulla; it is at the first vertebra, is it not?

DR. HANELIN: Yes.

The barium enema shows no evidence of intrinsic or extrinsic colonic abnormality. There is an interesting shadow visible in the right upper abdomen, which may be of importance. It is superimposed upon the liver and kidney and may represent the clinically palpable mass. Films of the upper gastrointestinal tract show no intrinsic abnormality of the stomach. The duodenal bulb, however, is deformed in a manner characteristic of chronic ulcer.

DR. JONES: In addition to the effect of surgical operation.

DR. HANELIN: It is difficult to be certain to what extent surgery contributes to this deformation. The second portion of the duodenum is displaced toward the midline and is indented along its lateral aspect. There is also a suggestion of a smooth pressure defect along the lesser-curvature aspect of the stomach.

DR. JONES: It could be the left lobe of the liver.

DR. HANELIN: Yes, that is its usual location.

DR. JONES: In other words, there is no evidence of a gastric lesion, which I think is of some importance. There is an abnormality here that has to be taken into account. It could be a gallstone that would have to be in the upper part of the common duct or, as Dr. Hanelin said, in the cystic duct near its junction but not near the ampulla. It would be high for that. This is the region of the first lumbar vertebra, and usually the ampulla is at the junction of the second and third lumbar vertebras. Is that correct?

DR. HANELIN: That is correct. A tumor of the head of the pancreas, if large enough, should encroach upon the second portion of the duodenum from its inner aspect.

DR. JONES: In addition, something of interest that does not help in the diagnosis is the appearance of the fine calcification in the region of the body of the pancreas. It may well be pancreatic calcification. It does not explain the entire history to me, although calcification of the pancreas may be associated with repeated attacks of pain, jaundice, fever and loss of weight. To be sure, this is the first attack, but I doubt very much if we could do more than say that there may be some disease of the pancreas that has caused calcification but is not the cause of the symptoms. In my mind, it has no significance as far as the presence or absence of cancer is concerned. If I understand Dr. Hanelin correctly and interpret these films correctly, there is no evidence of involvement of the duodenum by compression from a cancer of the pancreas.

DR. HANELIN: That is correct.

DR. JONES: The duodenal loop is not compressed from the inner side, suggestive of pancreatic disease. It might suggest something external to the duct or absolutely contiguous with the duct, possibly arising there or within the gall bladder. I should say that the suggestion that Dr. Hanelin made that there is some pushing of the duodenum toward the midline is much more evidence of tumor. It does not give any idea of the nature of the tumor. Therefore, I am left where I was before I saw the x-ray films. I believe he had a gallstone and some other disease in addition. I do not believe that pancreatitis alone will explain it. I would expect to find cancer, probably of the duct itself or of the ampulla, possibly of the gall bladder. I should be very much surprised if he had only stones in the common duct and nothing else. He certainly should have been explored in the hope that that might be the case and, lacking that, in the hope that cholecystenterostomy of some sort might permit palliation of a more serious condition and possibly resection of the tumor.

The other thing I did not comment on was the second x-ray finding of a nonfilling gall bladder. With jaundice it is not surprising that the gall bladder did not fill. With diminishing jaundice the result of the second Graham test should have been anticipated. Nothing was said about the stools. If bile were going through, some dye might have

seeped in, which surely suggests disease of the gall bladder.

Clinical Diagnoses

Cholelithiasis, with common-duct stone.

Peptic ulcer.

Dr. Jones's Diagnoses

Gallstones.

Cancer of biliary tract, probably of bile duct.

Anatomical Diagnoses

Adenocarcinoma, Grade II, of head of pancreas, with metastases to peritoneum, liver, gall bladder, regional lymph nodes, ileum, stomach and lung.

Chronic pancreatitis.

Ascites.

Common-duct stone.

Cholecystgastrostomy, recent.

Pathological Discussion

Dr. Tracy B. Mallory: At exploratory operation the peritoneal cavity was immediately found to be studded with cancer metastases, as was the surface of the liver. Because of adhesions in the region of the stomach and duodenum, it was impossible to carry out an extensive exploration, but the surgeon was under the impression that the head of the pancreas felt like cancer and on that basis did a cholecystgastrostomy.

Dr. Jones: Did he describe the gall bladder?

Dr. Mallory: It was distended. It is not stated to what degree, however. The patient lived only four weeks after operation. At the time of autopsy we found extensive cancer of the head of the pancreas and also very extensive chronic pancreatitis involving all the rest of the organ and, I think, explaining by means of fat necrosis the calcification that the x-ray examination showed in the body of the pancreas. There was a stone in the common duct a few centimeters up from the ampulla, as Dr. Jones predicted. The ampulla was quite free, for when the stone was removed, the common duct opened unobstructed into the duodenum. The jaundice, I believe, was due to the gallstone, not to the carcinoma. Between the time of operation and the autopsy the tumor had extended to some extent and now involved the serosa of the gall bladder and the stomach but was evidently not primary in either place. We could not find any trace of the old duodenal ulcer.

Dr. Jones: Was the liver enlarged particularly?

Dr. Mallory: Slightly, not greatly.

Dr. Hanelin: Was there any fluid in the lesser peritoneal sac?

Dr. Mallory: About 500 cc. in the general abdominal cavity, not in the lesser peritoneal space. There was a small, fluid-filled area immediately adjacent to the midportion of the pancreas in the retroperitoneal tissues. It contained only 30 cc. of fluid. There were also some carcinomatous implants on the serosa of the stomach.

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JUSTICE AND THE LAW

In science as in sociology the words of St. John: "And ye shall know the truth, and the truth shall make you free," have particular significance, for on them depends the direction in which mankind will move during the coming years. Massachusetts has now on the record two recent celebrated cases the final outcome of which will depend upon the degree to which truth can be served.

The first of these, the stand of the medical profession against the antivivisectionists, although still unsettled, has already spotlighted certain aspects of scientific progress. The second, Dr. Miriam Van Waters's fight for reinstatement as head of the Framingham Women's Reformatory, will, it is hoped, have educated the public in the ways of humane and progressive penology.

It is regrettable that public scrutiny should have been fixed on Dr. Van Waters's methods in such an atmosphere of malicious sensationalism. Whatever their motives in bringing the twenty-seven preposterous charges of "misconduct in office" against one of the foremost sociologists of our time, it seems evident that Commissioner of Correction Elliott E. McDowell and possibly others in penologic and political circles had a score to settle with Dr. Van Waters. Fortunately they failed; she stands completely vindicated, and her accusers are utterly discredited.

Parenthetically it may be added that not so many years ago Massachusetts lost the services of another able penologist, Howard B. Gill, who was ousted as head of the Norfolk Prison Colony, where McDowell was supervisor of workshops before he became commissioner last year. At that time, however, there was no state law giving public officials the right of appeal. Howard Gill stayed fired.

Dr. Van Waters served happily and successfully under five commissioners for sixteen years before McDowell was appointed. She filled well the shoes of such enlightened predecessors as Clara Barton and Mrs. Jessie Hodder. Her work has made Framingham one of the most studied and emulated penal institutions in the world.

Three months after he assumed office McDowell ordered the investigation of the reformatory. He had already indicated his hostility to her methods. The tactics employed during the inquiry were questionable. The charges revealed the spite and lack of vision of her accusers. They may be divided roughly into four categories, two of which may be dismissed without discussion.

One comprises accusations that Dr. Van Waters failed to stop sexual perversion among inmates. The review board appointed by the Governor found that everything possible had been done to contain a problem that exists in every situation where the sexes are segregated. The other includes a grab bag full of picayune charges, none of which were found to be just cause for removal.

These two groups of reasons seem to have been simply an attempt to justify the dismissal. They have little bearing on what appear to be the main

bones of contention. The other two strike closer to the meat of the matter.

One alleges that Dr. Van Waters failed to provide the prisoners with good examples by hiring former inmates as staff members. The Board called the charge "irresponsibly made" and added: "The example afforded by such persons, showing that it was possible to make good despite handicaps and errors, could be an important factor in the rehabilitation of other inmates."

The fourth, and possibly the most significant, group of charges accused Dr. Van Waters of violating state indenture laws by allowing inmates to be employed in other than domestic capacities. These laws were drawn up some seventy-five years ago. They specified that prisoners could work only as domestic servants. Since there were extremely few occupations open to a working woman in the 1870's, those laws are now inordinately restrictive. The board found that a fair amount of leeway in this respect had been allowed by former commissioners. It decided that the responsibility was theirs.

Commissioner McDowell once commented, "There can't be one law for great penologists and another for the rest of us." True enough. But laws are seldom changed until someone is courageous enough to test them and find them wanting. In a progressive society the letter of the law ever lags behind its spirit. Often enough, those who adhere too strictly to statutes whose origins are fundamentally humanitarian are left groping in the backwashes of progress with nothing to guide them but a dusty law book.

Clearly, Dr. Van Waters is ahead of the law. She has no use for the punitive aspects of prison life. She believes that enforced incarceration as an end in itself embitters more than it educates.

She calls her charges "students." Their troubles receive psychiatric attention, and the institution officers are their confidantes. They were encouraged to seek gainful employment in occupations of their choice and in line with their talents. Her entire program was geared to the principle of preparing the inmate for a successful return to the community.

She and others should be allowed to proceed on this principle. Nothing in the law should impede the temperate operation of the methods she has done so much to advance. Salaries should be raised to attract more persons of Dr. Van Waters's stature (both as institution heads and as commissioners of correction). The responsibilities of prison heads should be more clearly defined. The indenture laws should be liberalized. Even use of the term "indenture" — with its implications of bondage — might well be abandoned.

Let society catch up with Miriam Van Waters! That is indeed a challenge.

NEW PROGRAM IN PHYSICAL MEDICINE AT COLUMBIA

In May, 1944, Bernard Baruch made a grant of $400,000 to Columbia University to aid in the development over a ten-year period of a model center for teaching and research in physical medicine as recommended by the original survey by the Baruch Committee. Under the guidance of a committee on physical medicine of the medical faculty this project developed with signal advance in the therapeutic application of physical medicine, particularly including rehabilitation, and in education of technicians in physical and occupational therapy and of physicians training to specialize in the field. Some important basic research particularly in physiology has also been instigated.

This program has enlarged to such an extent that it includes the Columbia Presbyterian Medical Center, the Institute of Crippled and Disabled and eight other hospitals in New York City. In 1947 nearly 200,000 physical-therapy and occupational-therapy treatments were given in the medical center alone. To keep pace with this growth a new program has been announced by President Dwight D. Eisenhower. Dr. Robert C. Darling, associate professor of medicine at the College of Physicians and Surgeons, will serve as co-ordinator of physical medicine and rehabilitation, as well as director of research, a post he has filled for the past three years.

With the world-known rehabilitation facilities of the Institute of Crippled and Disabled and the wealth of clinical opportunities at Presbyterian and affiliated hospitals, together with a well co-ordinated research program, one should indeed anticipate at Columbia a model center for training and research in physical medicine.

Dr. Darling, the new co-ordinator, a graduate of Harvard Medical School, served as assistant director of the Fatigue Laboratory at Harvard and also as a consultant to the Quartermaster General. His studies have particularly dealt with various aspects of physical fitness, and he recently rendered a report on this subject in the *Journal of the American Medical Association* as chairman of the Baruch subcommittee on physical fitness. The organization of such a strong program in physical medicine at Columbia should attract competent young physicians into this rapidly growing and important field of medicine.

CANCER OF THE LUNG

OVERHOLT and Schmidt, in their paper on survival in primary carcinoma of the lung, delivered at the annual meeting of the New England Surgical Society last October and published elsewhere in this issue of the *Journal*, have made a valuable contribution to the increasing knowledge of cancer.

Improvement in the operability rates and in the operative results are encouraging but are to be expected, particularly in a branch of surgery that has been so recently developed as has been surgery of the lung. Less encouraging and presenting, indeed, a distinct challenge to both medical practice and medical public relations is the time that still elapses between the first symptom experienced by the patient and the establishment of the diagnosis.

This tragic interlude averaged eleven and three-fourths months for 133 patients who were seen between 1932 and 1942. Three months elapsed before the patient visited the doctor, another three months passed by before the first x-ray examination was made, and still another *five and three-fourths months* were lost before the diagnosis was made. Since 1942 this long interval has been reduced only to ten months. The doctor is now not consulted for three and eight-tenths months, the first x-ray examination is made one and six-tenths months later, and four and six-tenths more precious months elapse before the diagnosis is established.

This total interval can, conceivably, be completely eliminated by the same type of radiologic screening of the population that is now employed in tuberculosis case finding, for the growth can usually be discovered by the experienced radiologist or chest specialist before it begins to produce symptoms. Notice of abnormal areas of density spotted in survey films is reported back to the referring physician. It is his responsibility to see that any silent, abnormal density is properly labeled. His patient may have a silent, primary cancer of the lung, which, in most cases, is localized and can be treated successfully then, but not later.

Cancer of the lung should carry the highest rates of cure of any type of internal cancer because the time schedule of discovery can be set ahead to antedate symptoms. The responsibility is threefold: support by the laity of mass screening of the adult population on an annual basis; proper sorting of areas of silent abnormal densities by the medical profession generally; and reliance on exploratory thoracotomy in suspected cases.

UNWISE DRAFT DEFERMENT

ACCORDING to a recent release from the University News Office, the Faculty of Medicine of Harvard University has decided to continue its present policy of certifying for Selective Service deferment only students who are eligible to enter medical or dental school in the fall of the current year. Under the provisions of Local Board Memorandum No. 7 of National Headquarters, Selective Service System, the faculty was empowered to certify men in their first and second years of college by regarding them as premedical students and by provisionally recording their names for admission to future medical studies. The refusal to avail itself of this power represents a wise and honest decision on the part of the Harvard faculty.

A similar decision has been reached by a few other outstanding schools, notably, Boston University, University of Cincinnati, Johns Hopkins, University of Missouri, University of Wisconsin and Yale, but the remaining medical faculties throughout the country are still undecided. The dilemma confronting them is more important than it seems at first sight.

The folly of allowing military service to cut off the supply of future doctors, even in time of war, was

amply demonstrated by the sad fate of the Nazi Medical Corps, and there can be no quarrel with the deferment of men already embarked on a medical career. Whether we are fated to live through a long cold war, another hot war or no war at all, they will be needed. It was presumably at this point in their thinking that National Headquarters issued Memorandum No. 7.

But thinking need not stop there. The question is not the deferment of medical students but the deferment of "premedical" students. Granted that medical training is long, will a college freshman who is eighteen months older for having done his military service be, for that reason, deterred from following the career for which he believes himself best fitted? And, more important, can an eighteen-year-old boy be counted on to evaluate maturely his motives for calling himself "premedical" and accepting draft deferment? Certainly a youthful desire to escape the draft would be an inadequate and unfortunate motivation for the support of a life-long career in medicine — a career, by the way, that will not and should not exclude the possibility and the duty of later military service.

The decision arrived at by the Harvard Faculty is surely foresighted and sound, and it is hoped that a similar clarity of thought and sincerity of purpose will guide the rest of the medical faculties in resolving the serious issue that Memorandum No. 7 has placed before them.

MASSACHUSETTS MEDICAL SOCIETY

TREASURER'S REPORT COVERING REFUND DISTRIBUTION

The Treasurer of the Massachusetts Medical Society makes the following report regarding the refund to the district societies for 1949.

The Council voted to distribute the sum of $8000.00 to district societies. The total number of payments of annual dues received by the Treasurer by March 1, to be counted for the refund, was 4647. Therefore, the refund to the district societies for each paid fellow is $1.72.

The following table gives the number of payments, as of March 1, and the refund to each district as of March 9:

District	Number Reported Paid	Refund
Barnstable	44	$76.07
Berkshire	126	217.12
Bristol North	60	103.59
Bristol South	197	339.24
Essex North	181	311.72
Essex South	254	437.28
Franklin	45	77.79
Hampden	298	512.96
Hampshire	72	124.23
Middlesex East	132	227.44
Middlesex North	125	215.40
Middlesex South	936	1,610.32
Norfolk	870	1,496.80
Norfolk South	140	241.20
Plymouth	140	241.20
Suffolk	577	992.84
Worcester	362	623.04
Worcester North	88	151.76
	4,647	$8,000.00

In 1948, for comparison, the total number of payments for the refund was 4541.

Eliot Hubbard, Jr., M.D., *Treasurer*

THE $25.00 ASSESSMENT

On March 1, 1949, I sent the American Medical Association $50,000 as the first installment of the contribution of the membership of the Massachusetts Medical Society to the Campaign of Education. This represents payment of the assessment by about a third of our members. The checks are coming in to this office every day in a rather constant flow. One thousand checks were received within two weeks after the bills were mailed.

A description of the method we used here was sent to the other New England states and I have received word from them that they are all co-operating in this effort. The Washington State Medical Association has made the assessment compulsory, and I know of no other state society that has done so. Some states have increased their own dues for this public-relations effort. Michigan has done this for four years and has accumulated a fund of $400,000 to use in educating the public in that state concerning the nation's health.

I have received some checks for more than $25.00. A few checks have been for less than the amount asked because of personal financial difficulties. Only 12 fellows have written to say that they will not pay. Many more letters have been received praising this worthy effort.

It is my hope that by the end of March we shall have received checks from nearly 100 per cent of our members.

H. Quimby Gallupe, *Secretary*

DEATH

MEACHEN — John W. Meachen, M.D., of Boston, died on February 24. He was in his fifty-seventh year.

Dr. Meachen received his degree from Harvard Medical School in 1922. He was formerly a member of the staffs of the Massachusetts General Hospital, Cambridge Hospital, Free Hospital for Women in Brookline, Henry Heywood Memorial Hospital in Gardner, New England Hospital for Women and Children and Weymouth Hospital. He was a member of the New England Roentgen Ray Society and a fellow of the American Medical Association.

His widow, a son, a daughter, his mother, two brothers and two sisters survive.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATHS

DOLLOFF — Albert S. Dolloff, M.D., formerly of New Hampshire, died on March 6. He was in his eighty-seventh year.

Dr. Dolloff received his degree from Dartmouth Medical School in 1891. He was an affiliate fellow of the American Medical Association.

A son survives.

HART — Malcolm A. H. Hart, M.D., of Milton, died on January 24. He was in his eighty-eighth year.

Dr. Hart received his degree from New York University Medical College in 1888.

Two sons, three grandchildren and three great-grandchildren survive.

CORRESPONDENCE

DEPRIVATION OF LICENSE

To the Editor: At the meeting of the Board of Registration in Medicine held January 13, the registration of Dr. John P. Murphy, 129 St. Alphonsus Street, Roxbury, was suspended for three months.

GEORGE L. SCHADT, M.D., *Secretary*

State House
Boston

NOTICES

NEW ENGLAND CENTER HOSPITAL (JOSEPH H. PRATT DIAGNOSTIC HOSPITAL)

30 Bennet Street, Boston
Lecture Hall, 9-10 a.m.

MEDICAL CONFERENCE PROGRAM

Tuesday, April 5 — *Journal* Review.
Friday, April 8 — Some Further Notes on Auscultations of the Heart. Dr. Samuel Levine.
Wednesday, April 13 — Pediatric Clinicopathological Conference. Drs. James M. Baty and H. E. MacMahon.
Friday, April 15 — Fat in Parenteral Nutrition. Dr. Fredrick J. Stare.
Tuesday, April 19. Holiday — No conference.
Friday, April 22 — Some Observations on the Metabolic Requirements in Surgical Patients. Dr. Francis D. Moore.
Tuesday, April 26 — Newer Hematologic Methods for Studying the Blood Cells. Dr. Marvin Bloom.
Friday, April 29 — To be announced.

From 9 to 10 a.m. on Wednesday (except the second Wednesday), Thursday and Saturday mornings, clinics will be given by members of the hospital staff. Medical rounds are conducted each weekday except Saturday by members of the hospital staff from 12 to 1 p.m. On the *first* and fourth Fridays of the month, April 1 and 22, Therapeutic Conferences will be held with round-table discussion from 2 to 4 p.m., with Dr. Robert P. McCombs as moderator. On the *first* and fourth Fridays, April 1 and 22, Dr. Merrill Sosman will conduct X-Ray Conferences from 4 to 6 p.m.

On Saturday mornings from 9 to 10 a.m. Surgical Clinics are conducted by Dr. C. Stuart Welch.

All exercises are open to the medical profession.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, April 7, at 7:15 p.m., in the classroom of the Nurses' Residence, under the chairmanship of Dr. Ann P. D. Manton. Dr. Lorye E. Hackworth will speak on the subject "Congenital Intestinal Abnormalities in the Newborn."

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society will be held in the Lower Out-Patient Department Amphitheater, Massachusetts General Hospital, on Tuesday, April 12, at 8 p.m.

PROGRAM

Anatomy and Physiology of Splanchnic Pathways from the Viscera. Dr. James C. White.
Anatomy and Physiology of Pain Pathways in the Brain Stem and Spinal Cord. Dr. William H. Sweet.

A subsequent meeting will be held on May 10.

NEW ENGLAND DERMATOLOGICAL SOCIETY

The regular annual meeting of the New England Dermatological Society will be held in the Skin Out-Patient Department of the Boston City Hospital on Wednesday, April 20, at 2 p.m.

These meetings are open only to members and invited guests.

LONG ISLAND COLLEGE ALUMNI ASSOCIATION

The sixty-ninth annual dinner of the Alumni Association of the Long Island College of Medicine will be held at the Columbus Club, 1 Prospect Park West, Brooklyn, New York, on April 30, 1949, at 7 p.m. The principal speaker will be Dr. Jean Curran, president of the Long Island College of Medicine.

AMERICAN SOCIETY OF MEDICAL TECHNOLOGISTS

The seventeenth annual convention of the American Society of Medical Technologists will be held at the Hotel Roanoke, Roanoke, Virginia, from June 20 to 23. Further information may be obtained from Miss Ida L. Reilly, M.T. (ASCP), Roanoke Hospital Association, Roanoke, Virginia, the convention chairman.

VETERANS ADMINISTRATION RESIDENCY TRAINING IN NEUROPSYCHIATRY

A limited number of openings are available for July 1, 1949, appointment to the Veterans Administration residency-training program in neuropsychiatry. This program is under the jurisdiction of the deans of the Boston medical schools (Harvard, Tufts and Boston University). Training in this program, which may be from one to three years, is given at the following institutions:

Cushing Veterans Administration Hospital, Framingham, Massachusetts
Bedford Veterans Administration Hospital, Bedford, Massachusetts
The Mental Hygiene Clinic of Boston, Regional Office of Veterans Administration, Boston, Massachusetts
West Roxbury Veterans Administration Hospital, West Roxbury, Massachusetts
White River Junction Veterans Administration Hospital, White River Junction, Vermont

Emphasis in the entire program is on psychiatry with dynamic orientation and includes closed-ward, open-ward and outpatient care, child psychiatry and neurology.

All stations are within commuting distance of the Boston Psychoanalytic Institute, in the event that applicants have been accepted for analytic training.

Further information may be obtained from Dr. J. L. Hoffman, chief, Professional Services, Bedford Veterans Administration Hospital, Bedford, Massachusetts, or from Dr. Wilfred Bloomberg, chief, Neuropsychiatric Service, Cushing Veterans Administration Hospital, Framingham, Massachusetts.

(*Notices concluded on page xiii*)